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THE DIAGNOSIS AND TREATMENT  
OF  
DISEASES OF THE  
STOMACH AND INTESTINES

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## FOREWORD

To the man who practices medicine two questions arise with great frequency; what is the matter with my patient, and how shall I treat his ills? To answer these questions correctly is to practice medicine in a satisfactory manner. Consequently, diagnosis and treatment are of vital interest to every physician.

In the modern era of medicine very striking advances have been made in methods of diagnosis. Many new methods of precision have become available for bedside use. Discoveries in physics and chemistry have been applied in the clinic. Laboratory methods have been simplified so that they are available in diagnosis; many of them are such that they can be carried out readily by the physician himself. Older methods of examining the patient have been improved, and the implication of many signs long known are better understood. In utilizing these methods, even more than ever, is there need for clinical knowledge and judgment based on experience. The good diagnostician of today must know more facts, understand the use of more methods than was true of former generations of medical men.

For a time in recent progress in medicine it seemed as if advance in methods of treatment lagged behind advance in methods of diagnosis. There was a period of therapeutic nihilism. Many old methods of treatment had been discarded as better knowledge indicated their futility, and their place was not taken at once by more effective measures. In a period in

which pathological anatomy dominated medicine, and particularly a post-mortem pathological anatomy, effectively to treat many diseases seemed an impossibility. This period was followed by a newer pathology based more on a study of the living with advance in knowledge of function. Concepts of abnormal function stimulated renewed interest in methods of treatment. Great advances in our knowledge of etiology again brought improved methods of treatment along the lines of immunization, antitoxic sera, etc. Chemists developed a chemotherapy. Many new measures of applying physical methods in treatment were developed. A more rational basis for psychotherapy was evolved. From all of these changes there has come a present period of medicine in which treatment in all of its forms occupies possibly a larger place in medical literature than any other phase of medicine.

With so much known of diagnosis and treatment and with so many things being steadily added by medical investigators, there is a real need among those who practice medicine for comprehensive discussions of diagnosis and treatment by men competent to select what is best and to present it with sufficient detail and clarity for it to be of practical help in the problems of practice. For this purpose a series of "Monographs on Diagnosis and Treatment" has been prepared. Each volume is a complete discussion of the diagnosis and treatment of a certain group of diseases. It is offered with the hope that they, who read it, will be prepared to render to their patients a better service.

This volume by Dr. Cheney deals with diseases and disturbances of the gastrointestinal tract. It is striking how often patients complain of symptoms referable to the digestive tract; sometimes these are due to conditions within the digestive tract;

very often the main trouble is elsewhere. The physician needs to know where the trouble is and how to treat the situation. This is told in this volume.

HENRY A. CHRISTIAN, *Editor*.

PETER BENT BRIGHAM HOSPITAL

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PART I  
DISEASES OF THE STOMACH





## PART I

### DISEASES OF THE STOMACH

#### INTRODUCTION

Disturbed function of the stomach is very common. Dyspepsia, indigestion, "stomach trouble" are frequent complaints and constitute a large part of every physician's practice, but to decide what the symptoms mean is not always easy. All disturbance of function suggests organic disease, but not necessarily in the organ whose function is disturbed, and not inevitably in any organ at all, because faulty innervation from a number of different causes frequently upsets normal functions without structural change. Therefore, when a patient seeks relief from indigestion the problems presented are these: first, are the symptoms really due to disease of the stomach itself, and if so, what is the character of the disease? second, are they due to disturbance of the stomach's functions produced by disease in some other organ, and if so, where does the pathology lie? or third, are they altogether due to increased or decreased supply of nervous energy which deranges temporarily the stomach's sensation or secretion or motility? Upon the recognition and solution of these problems all successful treatment depends. But the determination of the truth sometimes presents great difficulties, and these stand out more prominently as experience grows. Consideration of the methods by which the physician seeks to distinguish one kind of disordered stomach from another and of those by which he endeavors to remove the disorder and restore normal performance of func-

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tion, is the subject of this book. Lengthy theoretical discussion has, so far as possible, been avoided, for explanation of any topic that will satisfy all observers and escape all contradiction is impossible. However, the statements herein expressed are based on experience and are believed to be trustworthy, and the entire object has been to give information that will be of practical value.

# CHAPTER I

## METHODS OF INVESTIGATION

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The methods available for investigation of a patient who complains of “stomach trouble” include clinical history, physical examination, gastric analysis, other laboratory tests, x-ray examination, gastroscopy and gastric photography.

### CLINICAL HISTORY

In every case of disturbed digestion, without any preconceived idea as to what the patient’s complaints may mean, a complete history should be obtained. This should cover all the various systems and not be limited to the gastrointestinal. For experience soon teaches that symptoms referred by the patient to the stomach may be the result of disease elsewhere in some other abdominal organ or even in some distant part of the body. Furthermore the history should include an account of the patient’s environment, habits of life, occupation and daily routine of work and relaxation as well as his past illnesses

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in addition to the present one. Only by such a complete history can one hope to attain a proper understanding of what the digestive disturbance means. A detailed account of the clinical history peculiar to each disease of the stomach is given later on in the consideration of that ailment, but this is never enough, and the clue to correct diagnosis may escape the investigator, if the whole story is not obtained. Of this fact the following is an instance: A woman, aged 53, complained of awful distress coming soon after eating for several months past, in spite of good appetite, with no other symptoms except much belching of gas. After all routine methods of investigation had failed to reveal any organic disease and the diagnosis thus remained indefinite, she confided that the onset of her digestive trouble had followed a quarrel with a gentleman friend with whom she had associated for years past and who had since bestowed his affections upon a younger woman. A nerve sedative soon gave relief to her indigestion.

### PHYSICAL EXAMINATION

Some of the most important diseases of the stomach, such as ulcer, may present no objective signs to identify them but only subjective evidence such as tenderness. Negative findings on physical examination are, therefore, never conclusive in gastric disease. On the other hand, careful inspection and palpation of the abdomen frequently prove of great assistance in the diagnosis. Even when examination of the stomach area reveals no sign of pathology, other parts of the abdomen or other organs outside the abdomen, when explored, may present abnormalities that explain the digestive disturbance. No part of the body, therefore, should be overlooked even though the patient's complaints concern only the stomach. The following

case exemplifies the truth of this: A woman, aged 48, complained that for a year past she had almost constantly "sour stomach" with intense burning appearing at a variable interval after eating. Physical examination of the upper abdomen was negative, but a supra-pubic mass was palpable, confirmed by pelvic examination. Her sour stomach entirely disappeared after removal of a large dermoid cyst of an ovary.

### GASTRIC ANALYSIS

The value of this in the diagnosis of disease of the stomach can not be emphasized too strongly, for it gives direct evidence as to how the organ is performing its functions of secretion and motility. However, it must also be recognized that the results obtained do not always mean organic disease of the stomach. Derangement of function, producing a secretion far above or below the normal average, may occur because pathology elsewhere has reflexly upset the stomach's work. Too much importance, therefore, must not be attached to the evidence obtained by this method of investigation, unless it is considered in connection with all other testimony elicited. Gastric analysis may be carried out in several different ways, each presenting both advantages and disadvantages, all of them more or less disagreeable for the patient, none of them perfect in details. The one longest employed is the Ewald test meal

1. *The Ewald Test Meal.*—The patient is seen in the morning while fasting, no food having been taken later than the midnight before. The Reh fuss or the Jutte or the mercury-weighted tube is passed, and the fasting stomach contents are aspirated for inspection, measurement and analysis, by means of a two or three ounce glass syringe. Then the tube usually

has to be withdrawn while the patient chews and swallows two slices of toasted bread and drinks a half pint of hot water. A half hour later the tube is passed again, and the first extraction of stomach contents is made for analysis. Four such samples are obtained in all, at half-hour intervals. The entire test thus requires about two hours. Such a procedure naturally has both advantages and disadvantages.

(a) *Advantages*. — The food taken is such as naturally would be eaten in the morning and constitutes a normal physiological stimulant to the stomach's functions. Toast particularly is appetizing to most people. It requires chewing before it can be swallowed, and so reflexly it excites digestive secretions. Furthermore, it possesses sufficient bulk so that it must be acted upon by the musculature of the stomach's wall to convert it into chyme. Thus its consistency when withdrawn affords evidence of the organ's motor as well as of its secretory power.

(b) *Disadvantages*. — The need to withdraw the stomach tube after the fasting contents have been obtained, in order to permit mastication and swallowing of the test meal in comfort, nearly always causes the average private patient to comment unfavorably when the tube has to be passed again for subsequent extractions. The usual duration of the examination, two hours as a rule, is for most people an ordeal, and for some it constitutes a serious loss of time. The technician, who conducts the test, frequently is annoyed by the thick character of the stomach contents, which block the tip or even the calibre of the small Rehfuß or similar tube, and always the samples removed have to be filtered before they can be analyzed. Finally the results obtained by the analysis have been questioned as not quite scientifically reliable, because the secretion is neutralized

to a certain extent by the cereal in the test meal and diluted by the water taken with the toast.

2. *The Alcoholic Test Meal.* — For this, in the same way as for the Ewald test meal, the patient is seen in the morning while fasting. The tube is passed and the fasting stomach contents are aspirated. Then, without removal of the tube, through the barrel of the syringe, after the plunger is removed, there are poured into the stomach two ounces (50 c.c.) of a 7 per cent. solution of alcohol in distilled water. Four extractions are then made for analysis at fifteen-minute intervals. This is found sufficient because with this type of test meal the stomach ordinarily is empty at the end of an hour.

(a) *Advantages.* — The stomach tube does not have to be withdrawn after the fasting contents are obtained and again introduced after the test meal is given. The whole procedure requires only one hour instead of two. Clear stomach contents are obtained that do not require filtration before analysis. Finally the results of the analysis as regards the stomach's secretory function are at least as accurate as those obtained by the Ewald meal, as repeated comparisons in the same patients have shown.

(b) *Disadvantages.* — The character of the test meal introduced is abnormal and does not represent a stimulant to secretion such as does ordinary food, for it lacks the psychic effects of savor and flavor and the mechanical effects of mastication. Most important of all, the alcoholic test meal furnishes no evidence as to the stomach's other important function, motility, for it does not require trituration, and the material extracted always is equally fluid.

In comparing these two different test meals, in the effort to decide which to recommend for routine use, it must be admitted

that the old and well-tried Ewald meal is as reliable as any and that in some respects it gives information even more valuable than that afforded by the alcoholic meal, because it tells us something about the stomach's motor as well as its secretory function. But from the standpoint of ease of performance and decreased discomfort to the patient the alcoholic test meal is to be preferred.

3. *Histamine Test Meal*. — The powerful action of histamine hydrochloride, when injected hypodermically, as a stimulant to gastric secretion has led to the suggestion that it be used as a routine test instead of the Ewald or the alcoholic meal. But the fact that its use produces at times transient symptoms that at least are disagreeable even though never serious, together with the fact that it constitutes a highly abnormal stimulant to secretion, in no way comparable to food, has made it seem undesirable to employ this method as a routine.

Histamine has been used for some time past, whenever complete achlorhydria was found by either the Ewald or the alcoholic meal, because after a small dose injected subcutaneously, one-quarter to one-half milligram, some stomachs react and produce free HCl even though they show none whatever after either of the other tests. In this way and by this time enough cases have been reported by different observers to make it appear that histamine alone can be used safely at the outset, instead of its employment only to supplement other tests.

As with either the Ewald or the alcoholic meal, the plan suggested is first to remove the contents of the fasting stomach as completely as possible; then with the tube still in place to inject subcutaneously one-quarter, one-half or even one milligram of histamine, the size of the dose depending upon the

size and weight of the patient. The exact dosage recommended is one-tenth milligram for each ten kilograms (22 lbs.) of body weight. Four extractions of stomach contents at ten-minute intervals are then obtained for analysis before the tube is withdrawn.

(a) *Advantages*. — The chief value of this method lies in the fact that it gives a pure juice, undiluted and uncontaminated by any test meal. The amounts obtained are entirely the result of the stomach's response to stimulation, unmixed with water or with alcoholic solution introduced with the test, and there is no possible neutralization of acidity by food. Thus both the quantity and the quality of the stomach's secretion are determined accurately, and by the repeated extractions throughout forty minutes evidence is obtained of this organ's ability to perform its secretory function. But the acid values obtained are decidedly higher by this method than by any other, and the figures must be expected to exceed those found by either the Ewald or the alcoholic meal.

(b) *Disadvantages*. — First, many patients in private practice object to the use of a hypodermic needle in carrying out any test, not only on account of the pain but even more because of the prejudice against this form of procedure. Second there is occasionally a painful local reaction about the site of the injection. Third, in at least one-third of the cases there is complaint of flushing of the face, a pounding of the heart, a sense of fulness in the head and of more or less headache for about ten minutes after the injection. Fourth, sometimes vomiting or diarrhea follows the use of histamine. Fifth, cases have been reported of severe shock, threatening collapse, but such an effect is rare, not exceeding one per cent. Sixth, most important of all, no proof is obtained by this method as to

how the stomach responds to the normal stimulus of food. By any ordinary test, no secretion might be found, and this evidence may be lost if histamine is used at the outset and not to supplement other tests only. At the present time, therefore, and at least until much more experience has been acquired with the use of histamine, it seems better to prefer other and longer-tried methods.

The material obtained by extractions from the fasting stomach and following a test meal should be examined in detail as soon thereafter as possible. This examination as regards the fasting contents, includes: (a) *gross inspection* as to quantity, consistence, general appearance, color, presence of food remnants, mucus, pus, blood and bile. The normal fasting stomach contents average 25 to 50 c.c. in amount, are liquid in consistence, slightly viscid, clear or pearly gray and opalescent, showing no sediment except possibly shreds of mucus. Fragments of food visible to the eye on direct inspection, such as bits of vegetable tissue or fruit-skins or grains of cereals or shreds of meat fibre are absolutely abnormal. Their discovery may mean only gastric atony but more often means pyloric obstruction of greater or lesser degree, depending upon the amount and character of the residue found. Fasting contents extremely thick and viscid, difficult to pour from one test tube or beaker to another, are characteristic of excessive mucus. Pus and blood, if present in quantity sufficient to be visible to the unaided eye, make the stomach contents appear thick and grumous, discolored a yellowish red and often offensive in odor. Blood alone in fasting contents imparts its characteristic color, from light to dark red. Bile is recognized easily by the green or yellow-green tint in the material removed. It does not belong in normal fasting stomach contents, and its

presence always signifies some pathology or abnormal function in the duodenum, which permits bile to regurgitate into the stomach.

(b) *Microscopic* examination of the sediment from fasting contents is accomplished easily by means of a smear made on a glass slide from the residue after filtration through sterile gauze. This smear may be inspected direct or better after staining with methylene blue. This may bring out retained muscle or vegetable fibres, otherwise indistinguishable. It may show red blood corpuscles even when there is not enough blood present to give color. It will identify pus corpuscles, if these are present. But the most common finding in the sediment from fasting contents is a quantity of micro-organisms, usually micrococci, swallowed during the night, from gums, pharynx or naso-pharynx, and of no significance unless unusually abundant. The long slender forms known as Oppler-Boas bacilli, occasionally found, are associated most often with gastric stasis from any cause, while the forms known as sarcinæ likewise are indicative of stasis and have no special significance. Rarely it is possible, in cancer of the stomach, to identify in the sediment tumor cells that are diagnostic.

(c) *Chemical analysis* applies to samples extracted after the test meal as well as to fasting contents. The material to be tested should be clear, and what is withdrawn must be filtered first, most simply through sterile gauze. The essential procedure is to determine the total acidity and the amount of free HCl. Total acidity is determined by taking a measured amount of the filtrate, preferably 10 c.c., to which is added a few drops of a one per cent. solution of phenolphthalein in alcohol, as an indicator. Then by means of a burette the filtrate is neutralized gradually by the addition of a decinormal

solution of sodium hydrate. This is allowed to run in drop by drop until a magenta color is produced, the end reaction being the point where the color does not deepen on the addition of more sodium hydrate. The amount of the latter required, measured in cubic centimetres, multiplied by ten, represents the total acidity. Free HCl is determined by adding to 10 c.c. of the filtrate a few drops of five-tenths of one per cent. solution in alcohol of dimethyl-amidoazobenzol (Toepfer's solution). This gives a bright red color, if free HCl is present. Then the decinormal sodium hydrate solution is added as before until the red color disappears and changes to a canary yellow. The amount of alkaline solution required to effect this change, as measured by the burette, multiplied by ten, represents the amount of free HCl.

Besides the tests for acidity, the benzidin test for occult blood should be a part of the routine, at least in the fasting stomach contents, even when on gross inspection no suggestion of blood is present. This test is carried out by dissolving a small pinch of benzidin in enough glacial acetic acid to effect its solution (one to two drams), and then adding an equal amount of hydrogen peroxide. To this test solution are added a few drops of the filtrate. The presence of occult blood is shown by a change of color to greenish blue or dark blue, depending upon the amount of blood present.

Bile usually is recognized easily in the gross specimens by its color without the necessity for chemical tests, but if it is desired to prove its presence, the nitric acid test (Gmellin's) is simple and readily carried out.

Lactic acid is recognized by taking 20 c.c. of a 1 per cent. carbolic acid solution, to which are added one drop of dilute ferric chlorid solution and sufficient water to form a transparent

amethyst-blue solution. If a few drops of the filtered gastric juice be added to 5 c.c. of this reagent, the solution will take on a canary yellow or greenish yellow color in the presence of lactic acid. This solution is not permanent and must be made fresh before each test.

All of these chemical tests are performed with little difficulty or loss of time; the information they give is vital to diagnosis, and they should not be omitted from the routine examination of any patient who presents the symptoms and signs of gastric disease. Those who have their patients in a hospital can get this work done in its laboratory. Those who maintain a laboratory in their office can have all the tests made by their own technician. But if such assistance is not available, any physician can easily learn to make these tests himself. The diagnostic significance of various findings obtained by gastric analysis will be discussed in connection with each disease in later chapters.

### OTHER LABORATORY TESTS

In every medical case certain laboratory examinations are indicated regardless of the nature of the history. Such are routine urinalysis, complete blood count and blood Wassermann or other form of test for syphilis, such as the Kahn, Hinton, etc. In certain special cases it becomes advisable to examine also the stool, to measure the blood urea, to investigate the spinal fluid after lumbar puncture and to test the basal metabolic rate. Disturbances of digestion not infrequently are a consequence of disease of the urinary tract, particularly of the kidneys, and urinary calculus may present symptoms simulating those of gastric ulcer. It is also a common observation that severe anæmia may suggest serious organic disease of the

stomach, such as cancer, and be distinguished from it only by blood examination.

A man aged 51 had been ill for a year with indigestion, loss of appetite, a sense of heaviness after food and gradual loss of weight. He was extremely pale but presented no signs of any gastric or other organic disease. Gastric analysis revealed complete achlorhydria. His blood count showed 38 per cent. of hemoglobin, his red cells numbered only 1,380,000, and smears showed a predominance of macrocytes. After intra-muscular injections of a solution of liver extract all gastric symptoms quickly disappeared with improvement in the blood condition.

As regards the Wassermann and allied reactions, disturbances of gastric function occasionally are a consequence of syphilis, even though syphilitic involvement of the stomach wall is rare. With pathologic lesions in the intestine, predominating symptoms may point to the stomach, but stool examination gives a clue that aids in correct understanding. Gastric crises may be suggested by signs such as abnormal reflexes, but examination of spinal fluid makes the diagnosis certain. "Nervous indigestion" sometimes means hyperthyroidism, and a test of the basal metabolic rate best reveals what causes the symptoms. Thus it becomes evident that to escape error thorough investigation is essential. The laboratory test, omitted because it seems unnecessary, may be the very one upon which accurate diagnosis depends.

### X-RAY EXAMINATION

Developments in recent years have proved more and more certainly that fluoroscopy and x-ray films are indispensable in the diagnosis of gastric disease. All other means of investigation lead only to inference; the films alone provide positive demonstration. Nevertheless there are limitations to be borne

in mind, if error is to be avoided. *First*, x-ray examination, particularly of the stomach, ought to be carried out only by experts who devote their time exclusively to this kind of work. By experience alone can the essential technique be acquired, by which abnormalities that exist are brought out properly, and defects that do not exist are not made to appear. Equally important is the proper interpretation of what is seen and the report that is sent out to the clinician. In any large city proper x-ray examination and intelligent report usually can be obtained at the various hospitals or at commercial x-ray laboratories. In smaller cities, likewise, physicians will get best results by leaving x-ray work to one or several among themselves who devote special time and study to it. Wrong conclusions are likely to mislead the man who does his own x-ray work in connection with all the other duties of his profession. *Second*, x-ray examination should never be the first step taken in the investigation of a patient as a short cut to diagnosis. It should always follow painstaking history, physical examination and laboratory tests such as gastric analysis, and its findings should be considered only as one link in the chain of evidence. No one method of investigation is infallible in gastric disease, and all the facts acquired by all methods combined are essential to correct diagnosis. It is a good rule to disregard x-ray findings that conflict with all other evidence, particularly if there is doubt about the quality of the x-ray report.

## GASTROSCOPY

Ever since Kussmaul in 1868 first devised an instrument to permit direct inspection of the stomach's interior, attempts have been made to gain information by this method of investigation. But Kussmaul's original instrument and all subsequent

modifications until recently have been in the form of a rigid tube like the œsophagoscope. This was always difficult to introduce and extremely disagreeable for the patient; occasionally it did harm to the tissues through which the instrument passed to reach the stomach, if force was employed rather than skill and dexterity, and after all, it gave a very limited explorable area. In the last few years, however, an entirely new form of instrument has been devised, that is flexible rather than rigid and gives a view of the stomach's interior, not by direct inspection, but rather by a system of lenses that deflect the image. This is known as the Wolf-Schindler gastroscope and has been in use since 1932. It is not very difficult to introduce; it does not disturb the patient to any extent, particularly if local anæsthesia of the pharynx and upper œsophagus precedes, as Schindler advises; it is much less likely to cause injury except by the most awkward manipulation. Furthermore it permits parts of the stomach to be visualized that previously could not be brought into view with the straight tube. The one advantage the open, rigid tube has over the flexible one is that it permits a bit of tissue to be removed through it for biopsy, and this can not be done with the Wolf-Schindler instrument. Much information has been acquired, however, by the new model that could not be obtained by the old, and reports by Chevalier Jackson in Philadelphia, Benedict in Boston, Schindler in Chicago and others indicate that this method of investigation will become more and more valuable as time goes on. It should be distinctly understood, however, that the gastroscope ought not to be used by the general practitioner without previous instruction and training in its introduction, by experts who have acquired the proper technique by experience.

## GASTRIC PHOTOGRAPHY

An instrument called the gastro-photor, invented by Back and Heilpern of Vienna, is now available for diagnosis but unfortunately at a price that is practically prohibitive for the individual physician. This instrument consists of a flexible rubber tube carrying the wires for electrical illumination and for operating the camera after it has been introduced into the stomach. The camera itself at the end of the rubber tube is about two inches long and a half inch in diameter and not flexible. It carries eight films so that eight views of the stomach can be obtained simultaneously, two in front, two behind and two at each side. It is less difficult to operate than the gastroscope and seems to be free from all danger to the patient. It is passed like any stomach tube after the stomach has been made clean and inflated with air. Only about thirty seconds are required for the actual photography. Valuable as seems the information that can thus be acquired, the instrument has so far had no general acceptance as a diagnostic aid.

CHAPTER II

ACUTE GASTRITIS

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How frequently this occurs is not easy to determine. Many patients with mild attacks may never call a physician at all. Others, who think they have this disease, present its symptoms only as one part of some other ailment much more serious. It is undoubtedly common, but usually it is brief in duration and occasionally so trivial in its manifestations that it receives no definite name.

Acute gastritis is caused most often by dietary indiscretions such as eating too much food at a meal, or eating food that is irritating or tainted or improperly cooked, or perhaps most frequently of all by drinking rather than eating to excess, ice-water, ice-cream sodas, iced tea, or alcoholic beverages of various kinds. Now and then an attack is due to some chemical irritant taken into the stomach by accident or intent.

CLINICAL HISTORY

As a rule the onset is sudden with a sense of discomfort in the epigastrium, a feeling of fulness and distension. Gradually this grows to an aching pain of greater or less degree. Then follows nausea and ultimately vomiting, which is repeated

until the stomach is emptied; even after no food remains, a thin fluid consisting of gastric secretion, mucus and regurgitated bile may continue to be evacuated. With these symptoms involving the stomach, headache and a sense of weakness and prostration occur but slight if any fever. Part of the offending stomach contents may pass on into the bowel before vomiting occurs and so add to the picture intestinal pains and diarrhoea. The whole attack usually lasts but one or two days, though it may leave the stomach irritable and resentful for several days more.

### PHYSICAL EXAMINATION

The tongue is coated, the breath offensive, the mouth dry. More or less tenderness is found over the epigastrium but not elsewhere over the abdomen. The temperature is normal or slightly above and the pulse moderately quickened. If there is fever of any consequence or the pulse rate is high, some other pathologic condition instead of acute gastritis should be suspected.

### DIAGNOSIS

A number of more serious ailments make their appearance at times under the guise of acute gastritis. Most notorious of these is *coronary artery occlusion*, with myocardial infarction. The newspapers not infrequently record the sudden death of some individual from "acute indigestion". In these cases the story is that, shortly after a full meal, the patient complained of a sudden pain in the upper abdomen, followed by vomiting and prostration, ashy pallor of the face and profuse sweating, only too often with death as a sequel at once or within an hour or two. The violence of such an attack, the rapid progress of

the symptoms, the evident presence of some emergency threatening life, with discovery of a weak, rapid, thready pulse, should make it clear that the condition is a much more serious one than simply acute gastritis. But the manifestations of coronary artery occlusion are not always so outspoken, and this possibility needs to be kept in mind in every attack supposed to be acute gastritis.

A man aged 62, a personal friend of the writer, presided one evening as toast master at a banquet given by medical men. Shortly after reaching his home about midnight he complained of pain in his stomach and vomited repeatedly. He was supposed to have an attack of acute indigestion as a result of his banquet. But he died within an hour, and the autopsy showed coronary artery thrombosis.

*Acute appendicitis* stands next in order of importance. This often makes its entry on the scene with a sudden gastric upset simulating acute gastritis, but the initial discomfort in the upper abdomen gradually shifts to the right lower quadrant and becomes more severe pain; emptying the stomach by vomiting does not give relief; slight fever at the outset persists and increases, and the well-known signs of tenderness, fulness and rigidity develop in the right iliac fossa. Rising pulse, rising leucocyte count with persisting pain, vomiting and fever prove within a few hours that more than the stomach is involved. A third stumbling-block in diagnosis, not so common as the two preceding but always likely to deceive is the *gastric crises* of *tabes dorsalis*. The characteristic features of such an attack are vomiting and pain, sometimes one, sometimes the other predominating. Their onset is sudden and their duration usually brief. They leave the patient as well afterwards as before. The abdomen presents no physical signs except epigastric

tenderness. The most suggestive point about the attack that makes the diagnosis of acute gastritis dubious is its violence and the extreme severity of both the vomiting and the pain. The clue to diagnosis is found in the disturbance of superficial reflexes, particularly pupillary, patellar and Achilles, and in peculiar features of the spinal fluid obtained by lumbar puncture. As a matter of fact, the first gastric crisis probably is seldom recognized for what it is but is set down as acute gastritis. With repetition of the attacks some other explanation seems more likely, and then with more complete examination the true condition becomes evident.

A man aged 36 was seen at Lane Hospital complaining of violent pain in the pit of his stomach and excessive retching and vomiting for twenty-four hours preceding. One month before he had a similar attack but meantime had been well. There was no fever, pulse was normal, abdominal examination negative. In two days again all symptoms had disappeared. But it was found that both pupils were small and fixed and failed to respond to light. Both patellar and both Achilles reflexes were entirely absent. Spinal fluid by lumbar puncture showed 55 cells; positive Wassermann reactions and typical colloidal gold curve.

Other occasional sources of gastric attack resembling acute gastritis are *uræmic poisoning*, identified by urinalysis, blood pressure, high blood urea; *early pregnancy*, recognized not only by history and pelvic examination but also by the Aschheim-Zondek test of urine; and the onset of various *acute infections*, such as pneumonia, meningitis and malaria, each of which presents signs of its own, in addition to gastric disturbances. Thus the diagnosis of acute gastritis should always be made by exclusion and not accepted until routine methods of investigation previously described have eliminated more serious possibilities.

## TREATMENT

Once certain that the condition is really acute gastritis, the less interference the better. Many a time irritation of the stomach is kept up by drugs administered to relieve it. The patient should be placed at rest in bed. No food should be given for hours after the onset, perhaps not for one or two days. For thirst, small amounts of albumen water may be taken every hour or two, but not over one-half ounce at a time; this albumen water is made by dissolving the white of one egg in eight ounces of cold water. Alkaline mineral water such as Vichy Celestin may be substituted for or alternated with the albumen water, in similar amount.

No other remedy gives relief so quickly to vomiting and discomfort as lavage to remove offending stomach contents. The best solution to employ is bicarbonate of soda one heaping teaspoonful to a pint of comfortably hot water. This procedure, however, occasionally will not be tolerated by the patient and force should not be used.

A mustard paste made from one tablespoonful of powdered mustard with three tablespoonfuls of white flour, spread on cheese cloth or thin linen or sterile gauze and applied over the epigastrium as a counter-irritant, serves likewise to relieve discomfort and nausea. It should remain until the skin is thoroughly reddened; then should be removed from the front of the body and applied in the same way over the back, opposite the site already treated, again until there is definite erythema produced but not until blistering results.

No drugs are of much benefit, but if a prescription is desired, the best gastric sedative is calomel, given in dose of 15 mgm. (gr.  $\frac{1}{4}$ ) or less with 60 mgm. (gr. 1) of bicarbonate of soda,

every half hour in a teaspoonful of water until twelve such powders are administered. This may be followed by one or two teaspoonfuls of milk of magnesia every hour until the bowels move freely.

Feeding should be resumed slowly, liquid at first exclusively with small amounts of any clear broth, cereal gruel or malted milk given every two hours. Gradually these quantities may be increased and toast, crackers and well-cooked cereals may be added. No attempt should be made to return to a regular diet, unrestricted in quality and quantity, for at least one week. Five drops of tincture of *nux vomica* in a tablespoonful of water three times a day will aid the stomach at this stage to recover its normal motility, usually impaired by the acute attack.

CHAPTER III

CHRONIC GASTRITIS

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In the past the very existence of this form of disease of the stomach has been questioned by authorities. Patients rarely die from it, so that the diagnosis has not been subject to review by autopsy; surgeons are never led by the symptoms it presents to open the abdomen, so that opportunity has seldom been afforded for direct inspection of the gastric mucosa during life, to prove or disprove the reality of this disease; there has been until recently no characteristic picture presented by x-ray films to identify it. Without these checks to inaccuracy, the tendency has been either to discard chronic gastritis entirely from the list of recognizable ailments, or else to include under this designation a number of conditions of uncertain pathology that have cast discredit upon the diagnosis and have even caused it to be branded “a cloak for ignorance”. However, within the past few years, since the invention of the Wolf-Schindler gastroscope, a great deal of attention has been devoted to the subject of gastritis and numerous descriptions have been

published of the appearance that different pathological types present under direct inspection. Also the x-ray experts have developed a special technique to bring out abnormalities in the gastric mucosa; so that the fact that there is such a disease as chronic gastritis has been established beyond question.

The causes of the disease are important from a diagnostic standpoint, because their discovery in a patient's history or physical examination aids in a proper interpretation of the symptoms presented. These causes can be classified under three heads: (1) *chronic irritation* due to habitual indiscretion in the taking of food and drink, as regards quantity, quality, seasoning, temperature, trituration and the regularity of meals; (2) *chronic infection*, from the constant swallowing of pus or micro-organisms from foci in gums or teeth, particularly from pyorrhea and pyorrhea pockets, or from diseased sinus or tonsils, or from trachea or bronchi; (3) *chronic congestion* in the stomach's walls, due to portal obstruction from hepatic disease, or to circulatory stasis from myocardial insufficiency. Any one of these factors may act alone, or any two or all three may act together.

### CLINICAL HISTORY

The disturbance of function produced by chronic gastritis gives rise to varied complaints; so varied, in fact, that from them alone no proper conclusion can be reached. This is where error has arisen in the past, for all sorts of disorders of digestion, judged by their history only, have been assigned to the group without adequate proof that they belonged there. The complaint most often made is of discomfort coming soon after eating or even while eating, variously described as fulness, distension, a sense of weight or a load, sometimes as a dull ache, but never

as severe pain. This disagreeable sensation always appears promptly after food is taken, not after one to four hours of comfort as in gastric ulcer or in hypersecretion from some extra-gastric cause. Next in frequency is complaint of belching, of repeated eructations of gas, continuing at intervals for a long time after meals and gradually giving relief from the sense of distension. Compared with these two symptoms, distress and gas, all others are much less constant. Some patients complain also of nausea habitually after meals. Sometimes this persists until vomiting occurs, with evacuation of the food just eaten and of thick slimy mucus. Some feel nauseated only when they rise in the morning and must then strain and retch until they bring up a little viscid mucus. Some induce vomiting regularly for the relief it affords to their discomfort. Appetite is not always poor; often it is said to be good, but patients dread to eat because of the distress food causes. Complete loss of appetite is unusual. Loss of weight is exceptional, and fair nutrition is maintained as a rule, no matter how much complaint is made of "dyspepsia". Heartburn in some cases is a prominent and constant symptom, with eructations at times of a mouthful of food or of hot, bitter, irritating fluid, when gas is expelled from the stomach. But ordinarily "sour stomach" does not occur in this form of gastric disease.

Reflex manifestations are common in chronic gastritis and may form so large a part of the history that they distract attention from the stomach itself. The most important of these is headache, occurring either in the morning when the patient wakes, or after meals with the other symptoms described, or only at intervals of days or weeks, lasting for one day with vomiting and again disappearing. The pain usually is a dull ache or sense of heaviness, felt across the forehead and over

the top of the head, but it may be extremely severe and prostrating as in "sick-headache" attacks with vomiting. Other reflex symptoms are drowsiness after meals, lack of energy at all times and indisposition for exertion, dizziness, and particularly depression of spirits and irritability of temper.

All the symptoms described may persist over long periods, for months or years, at times better, at times worse. There is nothing about them to alarm the patient, such as severe pain, emaciation, vomiting of blood, or any other event that gives the impression of danger to life. Therefore, many individuals go on indefinitely, enduring the inconvenience and annoyance that their chronic gastritis causes, treating themselves by digestive tablets or by one fad or another, before they come at last to seek medical advice. They gradually acquire the habit of self-examination and introspection, so that they are eager when questioned to give an elaborate and detailed account of their numerous ailments. These peculiar features of long duration of symptoms, variability in their character and severity, fairly good nutrition in spite of constant disturbance of digestion, the tendency to periods of improvement and of exacerbation but never progressing to fatality or to complete recovery, all help to identify these patients with chronic gastritis, but never are sufficient to establish the diagnosis.

#### PHYSICAL EXAMINATION

This gives little help. No sign is found with any frequency except tenderness over the epigastrium, and this is by no means constant. A succussion splash over the stomach, hours after food was taken, indicates a weak wall and slow peristalsis, but it does not tell why. The evidence is all negative; no tumor, no peristaltic wave, no bulging, no rigidity over the gastric

area or elsewhere. The finding of foci of infection in the mouth, throat or sinuses, or of a moderately enlarged liver, or of an inefficient heart, strengthens the suspicion that the disturbed gastric function means chronic gastritis, but it does not prove it.

### GASTRIC ANALYSIS

This part of the examination and this alone supplies the evidence on which the diagnosis depends. (1) The fasting contents are not large but consist mainly of viscid mucus. Sometimes remnants of food are found, visible to the naked eye, but more often requiring the microscope for their detection. In the smears from the sediment, muscle fibres, starch granules, vegetable debris, yeast cells and sarcinae are common findings; occasionally red blood cells are seen, and a variable number of micro-organisms, very abundant in the focal infection types, together with pus corpuscles and desquamated epithelium. (2) After the Ewald test meal, fractional analysis shows poor trituration, deficient secretion as proved by the thick, sticky character of the material recovered, at all extractions an excess of mucus, mixed with the food or separate from it, while the total acidity is low and the free HCl deficient or absent throughout the examination. It is not uncommon in these cases to see a delayed secretion, slowly rising and highest two or two and a half hours after the test meal is taken, but even then not reaching the average normal. It is also common to see a discrepancy between the total acidity and the free HCl, the latter remaining low or absent, even though the former may approach the normal.

It is the combination of poor trituration, as shown by inspection of the contents removed, slow peristalsis as demonstrated by the presence of food fragments from a previous meal,

scanty secretion of gastric juice, as proved not only by the small amount of material obtained and the lack of fluid in it, but also by the analysis, with the abundant mucus in fasting contents and in all samples during digestion, that forms the characteristic picture in chronic gastritis. Some of these features may arise from other causes, but mucus in abundance is the product of chronic gastritis only and the sign that makes the diagnosis certain.

### X-RAY EXAMINATION

This supplies no direct proof of chronic gastritis by methods ordinarily employed but is nevertheless of great value because it eliminates other forms of organic gastric disease. It may reveal a large stomach with feeble peristalsis and slow emptying time, but without pyloric obstruction to account for the delay. Such a dilated atonic organ is commonly the result of chronic gastritis, so that its discovery is suggestive, but it is not conclusive, for it is not always shown by x-ray examination, even when history and gastric analysis combine to make the diagnosis reasonably certain, and it may be found without chronic gastritis, due to a gastric neurosis affecting motility. It is possible now, moreover, for the expert in x-ray technique, to demonstrate the presence or absence of abnormalities in the mucous membrane lining the stomach and to bring out features characteristic of chronic gastritis. This is done by special methods concerned especially with the kind and amount of paste administered for visualization.

### GASTROSCOPY

Much attention has been paid to the investigation of gastritis since the Wolf-Schindler gastroscope became available,

and much information has been added about the changes visible in the stomach's mucous membrane in chronic gastritis and the different appearances in different forms. An entire book on the subject of gastritis alone, acute and chronic, has been published in Germany by Henning, based largely upon the recently acquired knowledge made possible by gastroscopy. As time goes on and the gastroscope is more commonly used, this method of investigation undoubtedly will become the court of last resort in identifying the group of diseases long known as chronic gastritis and in segregating them from others less or more serious.

### DIAGNOSIS

Even though the general practitioner does not have at his command the services of a trained gastroscopist and an expert x-ray technician, usually it is possible for him to reach an accurate diagnosis of chronic gastritis by older and simpler methods. The typical case of chronic gastritis is known by the following features: a history of habitual distress after meals with belching of gas, poor appetite, nausea, vomiting after eating or in the early morning after rising, dull headache with drowsiness while digestion is in progress, depressed spirits, querulous disposition and a tendency to tell a long tale of unimportant minor ailments; negative findings on physical examination or perhaps tenderness over the stomach or a succussion splash; the finding by gastric analysis of deficient secretion, poor trituration and mucus in excess and a stomach normal in contour shown by x-ray films, or possibly with walls weakened and over-stretched that act slowly in consequence.

The first problem presented is to sift out the cases of real organic disease of the stomach characterized by chronic inflam-

mation of its lining membrane, from the mass of functional disturbances of digestion, the so-called *gastric neuroses*. The one reliable dividing line between the two has always seemed to be the formation in the stomach of abundant mucus, as shown by the gastric analysis. The secretion may be subnormal in amount and acidity, and the motility may be poor in patients with depression of nervous energy, or the secretion may be above normal in amount and acidity and the motility exaggerated in patients with excessive nervous excitability, but the presence of thick, stringy mucus in abundance in fasting contents and after a test meal usually is reliable evidence of gastritis, for it does not occur in purely nervous disturbances. If doubt still remains, direct inspection by gastroscopy will settle the question.

The history and signs of some adequate cause for chronic gastritis afford further testimony to sustain its diagnosis. For instance, a confession of habitual over-indulgence in the pleasures of the table for a period of time preceding the onset of symptoms of indigestion naturally creates a suspicion that chronic irritation may have resulted in chronic inflammation. A story of long-standing infection in gums, teeth, tonsils or a sinus with the constant swallowing of infectious material justifies the conjecture that the patient's ultimate development of digestive disturbances may be due to chronic gastritis as a sequel. If a weak myocardium with inadequate systolic output is discovered in a patient who seeks advice for stomach trouble, it is reasonable to infer that circulatory stasis in the gastric wall may be responsible for the symptoms and for the signs found on gastric analysis. Finally, when changes in the size of the liver and possibly of the spleen are noted in the course of a routine physical examination, it is probable that the

digestive disturbances are again due to circulatory stasis in the stomach's walls as a consequence of chronic portal hepatitis. In such instances the chronic gastritis is of course not the primary disease but only one part of a larger picture.

Another interesting problem concerns the relation of chronic gastritis to *chronic cholecystitis*. There can be no question about the fact of the association of the two, though there is about the frequency with which this occurs. Statistics vary but the coincidence certainly exists in from one fourth to one half of all the cases of chronic inflammation of the gallbladder. The finding, therefore, of achlorhydria or marked deficiency of gastric secretion after a test meal with mucus in excess in all extractions always should excite the suspicion that chronic inflammation of the gallbladder may hide behind the obvious stomach disturbance. Other evidence that implicates the gallbladder must then be sought in the history, by palpation of the right upper abdominal quadrant and by x-ray films after the dye has been given for visualization. But just as chronic gastritis can not always be interpreted to mean that chronic cholecystitis is its cause or associate, so when cholecystitis is diagnosed, it does not always follow that chronic gastritis will be found accompanying it. The association is common but not inevitable. Why it occurs has never been adequately explained.

A woman, aged 60, first seen in 1915, complained chiefly of "stomach trouble". The symptom that annoyed her was "a heavy aching" coming on in the stomach after eating with belching of gas but no vomiting. This had gone on more or less constantly for years past. No abnormality was found on physical examination of her abdomen except tenderness over the right upper quadrant. After the Ewald test meal there was found an abundance of thick mucus, poor trituration of the toast, very little gastric secretion and no free HCl. X-ray films showed no abnormality in the contour

of the stomach and no shadow of gall-stones in the flat plates. This patient was under observation at intervals for four years and treated during that period for chronic gastritis. Repeated gastric analyses always showed the features described. As time went on she began to have intercurrent attacks of pain in her right side at the costal margin, a dull ache lasting for a few days, and gradually these became more frequent and more violent. As no plan of treatment cured her indigestion, she was finally persuaded to have her gall-bladder removed. This was done in 1919. The walls of the organ were found thickened and tough, and one large round stone occupied its entire lumen. This patient was last seen for another ailment two years after her operation and reported then she had had no further stomach trouble following cholecystectomy.

Now and then the manifestations of *chronic nephritis* resemble those of chronic gastritis so closely that they convince the patient the stomach is the cause of them all. The physician too may be misled unless all means of investigation are employed before a diagnosis is reached. Discomfort after taking food, nausea and frequently vomiting after a meal, dull headache more or less constantly, combine to call attention to the digestive tract, and no other symptoms may be prominent enough to suggest pathology elsewhere. But arterial hypertension, urine showing albumen and casts and blood containing an abnormal amount of urea usually suffice to show where the trouble really lies.

A woman, aged 44, sought advice for stomach trouble characterized by nausea and vomiting. For six months she had been vomiting after her meals at least once every day. She had no pain, but within an hour after eating she became nauseated and distressed, and some days she vomited every meal taken. She had no other complaint to make except an occasional "sick headache". Her face was pale, puffy and waxy in appearance. Her blood pressure was 210 systolic and 120 diastolic. Her urine showed a heavy cloud of

albumen but no casts. Her blood urea was 78 milligrams per 100 c.c. of blood. Her blood count was remarkably low, with 55 per cent. hemoglobin and only 2,310,000 red blood cells. In spite of all treatment this patient continued to vomit at intervals until her death a year later from progressive failure of renal function.

In the background of every picture of chronic disorder of the stomach stalks the spectre of *cancer*. In any patient with a history of persistent disturbance of gastric function, particularly if loss of weight, strength and color form a part of the story, cancer of the stomach should be the first thought. Absence or deficiency of free HCl in the stomach contents, poor trituration of food and abundant mucus may be found in either chronic gastritis or cancer. Never should the discovery of a palpable tumor be awaited to make the diagnosis between the two. X-ray films and fluoroscopy become in such a case indispensable aids in differentiation, but even they may fail at first to reveal the presence of a small, flat growth in the stomach wall. If this examination, however, shows no neoplasm, it is usually safe to conclude that none exists and that the condition is chronic gastritis only. But final decision should be delayed temporarily for further observation and for therapeutic test; if after one month no improvement has resulted, exploratory laparotomy had better be advised. It is in these doubtful cases that gastroscopy will prove of the utmost value, permitting direct inspection of the stomach's interior without preliminary abdominal incision to make possible examination of its walls.

### TREATMENT

The first indication is to regulate what goes into the patient's stomach. Definite instructions must be given about the diet,

as regards quantity, mode of preparation, consistence, temperature, composition and purity. It does no good to tell patients in a general way what to eat and what not to eat. They either forget or they do not heed the advice. At the risk of appearing dogmatic it is better to furnish them with a list to follow and to insist that they be guided by it. The following instructions for patients with chronic gastritis will be found satisfactory. They can be modified or supplemented from time to time according to developments:

“In general, food should all be soft and without tough particles or coarse fibre that will irritate the stomach. This includes *liquids*: (a) all clear soups, such as beef, mutton or chicken, cooked with rice, barley or vegetables, but strained; (b) all cereal gruels, made from barley, rice, farine, oatmeal or cornmeal; or from Eskay’s food, Imperial Granum, or Nestlé’s food; (c) peptonized cow’s milk, prepared by adding one-half of a Fairchild’s peptonizing tube to one-half pint of milk fifteen minutes before it is taken and warming immediately before drinking; (d) Horlick’s malted milk mixed with hot water according to directions on the package; (e) cocoa, made with Phillips’ digestible cocoa and cow’s milk, according to directions on the package; (f) bottled fruit juices, such as grape or pineapple; *avoid* coffee, tea and all alcoholic drinks. *Cereals*: such as farina, rice, germea, cream of wheat, cornmeal, oatmeal, provided each is cooked for three hours before it is eaten, served with a small amount of cream and sugar and always taken in moderation. *Bread-stuffs*: such as stale bread (at least one day old), toast, the crust of rolls, shredded wheat biscuits, triscuits, zwieback, water crackers; *avoid* all hot biscuits, waffles, hot cakes and any bread freshly baked. *Meats*: such as roast beef or mutton, beef steak or mutton chops, chicken broiled or roasted, provided the meat is tender, is not cooked too done so that it is hard, and is taken not more than once each day; *avoid* veal, pork in any form, all boiled or stewed meats and all fat. *Fish*: any variety may be eaten except salmon; broiled or baked, or boiled, never fried; *avoid* all salt, smoked and pickled fish, such as mackerel,

sardines and herring. *Shell-fish*: fresh oysters may be taken prepared in any way except fried; *avoid* clams, lobster, crab, shrimps and canned oysters. *Vegetables*: any of these is best prepared in the form of a purée, made by boiling thoroughly and then rubbing through a purée sieve, adding the purée to milk to make a cream soup or eating it separately as desired; the flower of cauliflower but not the stalk, the tips of asparagus, the centre of the artichoke, young and tender peas or string beans may be taken in moderation without preparation as a purée; potatoes should be eaten only when baked or thoroughly mashed. *Salads*: crisp lettuce, raw tomatoes, tender cauliflower, hearts of artichoke or any soft vegetable may be taken in the form of a salad, made with French dressing of oil, vinegar, salt and pepper; but *avoid* salads made with shrimps, crab, and lobster; and mayonnaise or other dressings. *Eggs*: eaten soft-boiled, poached or shirred, scrambled or in omelet always cooked so as not to be tough never hard-boiled eggs. *Fruits*: peaches, apricots, pears, figs or grapes may be eaten raw with the morning or noon meal, but never between meals; strawberries, blackberries, raspberries or loganberries should never be eaten, either raw or cooked, but their juice may be expressed, before or after cooking, and taken as a drink; orange juice or grape fruit juice may be taken similarly, but avoid the pulp of these fruits; baked apples or apple sauce is allowed but not raw apple; avoid bananas and all preserved fruits. *Desserts*: all pies, cakes, pastries, puddings, candies, ices and ice creams must be absolutely left alone.

Water may be taken freely between meals but never during the meal. Take food always in moderate amounts; take no food between meals; and above all else, chew solid foods thoroughly until well softened”.

All food enters the stomach through the mouth, and good material may there be poorly treated or even contaminated, so that it enters the stomach as improper material. Teeth play a most important part in the preparation of food for the stomach. If grinders are missing, solid substances can not be

trituated and softened as they ought to be before they pass on, and the same is true, if teeth are present but are not thoroughly used. Thorough mastication is essential to good digestion, and if people have not learned this sooner, they must be taught it when they acquire chronic gastritis. It becomes the duty of the physician, therefore, to see that the patient has teeth and that they are used properly.

It is almost equally important to be sure that the mouth is clean. Dental caries with cavities containing foul material, or disease of the gums around the tooth sockets or pyorrheal pockets between the teeth, lead to the constant admixture of micro-organisms with the food and with the saliva even when no food is taken; and these find their way into the stomach to act as bacterial irritants. The physician's most valuable ally in the treatment of chronic gastritis is the cook, but almost equally important is the dentist, and without the intelligent co-operation of these two as well as of the patient himself, all dietetic and medicinal means employed may fail to cure.

The objects of direct treatment are first to keep the stomach clean, and second to overcome impairment of its function caused by the inflammation of its wall. In years past it was taught that all these patients needed lavage, and the daily use of the stomach tube, even for months at a time, was part of the routine management. No doubt this method often was beneficial, but it was carried to extremes and has now gone out of fashion. Nevertheless in some cases with morning accumulation of mucus with nausea and retching on rising, lavage is a very useful procedure. It should be done before breakfast with a warm solution of bicarbonate of soda, one teaspoonful in a pint of water. Most patients can be taught quickly to do this

without the physician's aid, and in calculating how it benefits, the psychological effect must not be left out of account. In ordinary cases, however, and for the average individual the same result is accomplished, of cleansing the stomach of mucus and debris accumulated over night, by drinking each morning a half pint of hot water in which is dissolved a heaping teaspoonful of sodium phosphate. Whether this is vomited or passes on into the bowel, it has the same good effect. It should be taken about a half hour before breakfast.

To stimulate secretion and motility, impaired by chronic gastritis, bitter tonics before meals deserve first consideration. The best one of these is tincture of *nux vomica*, which may be given alone, in dose of ten drops in a tablespoonful of water before each meal. Instead of this, a combination of equal parts of tincture of *nux vomica* and tincture of *physostigma*, twenty drops before each meal, may be prescribed; or tincture of *nux vomica* and fluid extract of *hydrastis* of each five drops with fluid extract of *condurango* ten drops; or a mixture of tincture of *nux vomica* two drachms (8 c.c.), with compound tincture of gentian two ounces (60 c.c.) and compound tincture of cinchona sufficient to make four ounces (120 c.c.), of which the dose is one teaspoonful in water before each meal.

Alternating with this course of bitter tonics or prescribed coincidentally with it, dilute hydrochloric acid after meals is indicated to supplement deficient secretion. This may be given alone, in dose of ten or twenty drops well diluted in water, repeated at intervals during digestion if discomfort persists. Sometimes a combination with pepsine is more efficacious than the acid alone, in spite of the fact that pepsine usually is secreted by the stomach even when acid is not. Thus a dose of ten drops of dilute hydrochloric acid in a drachm (8 c.c.) of

Fairchild's essence of pepsine or gastron frequently gives more comfort than the acid by itself.

In every case of chronic gastritis something else is needed besides dietetic and medicinal therapy. These patients have usually acquired an abnormal outlook on life as a consequence of their persistent dyspepsia and frequently have become self-centred, moody and despondent. Their thoughts must be directed away from their ailments. For this purpose some need outdoor sports, such as golf, tennis, walking, horseback riding or motoring. Others obtain diversion more pleasantly by indoor amusements such as theatres, concerts or moving pictures, while all should be encouraged to take up some useful occupation, if they do not already have one. Finally, all these patients can be helped by psychotherapy along the lines of encouragement, cheerfulness and sympathetic reassurance on the part of the physician. This method of treatment has been allowed to pass too much into the hands of the ignorant, or of religious cults of one kind or another, but it belongs to the physician and he should not omit it from his resources.

# CHAPTER IV

## ULCER OF THE STOMACH

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Ulcer is the form of gastric disease most frequently seen. It is also the most persistent and the one most likely to recur, no matter what plan of treatment has been employed for its cure. It is always annoying, to say the least, and more or less incapacitating, and it leads to numerous complications that are threatening to life. Now and then it causes sudden death. For all these reasons ulcer of the stomach has received an enormous amount of attention and has been made the subject of almost constant investigation and discussion. And yet even now there is no general agreement as to what causes it nor as to the best method of treatment in order to effect a permanent cure; while many phases of its history, its effects and its management are a continual source of controversy.

There are certain accepted facts, however, that are essential to an understanding of the symptoms it produces, its protracted clinical course, its many complications and its occasional

disastrous outcome. For instance, the great majority of ulcers of the stomach are situated at the pyloric end; the ulcer may actually lie in the pyloric canal, though this is unusual; even more frequently it is found just beyond the pylorus, not in the stomach at all, but in the duodenum, within its first inch or the so-called "duodenal bulb". In the stomach the ulcer may be situated, not at the pylorus, but on the lesser curvature, two to four inches away from the outlet as a rule, but exceptionally even further along. It is rare to find one at the cardiac end or fundus or along the greater curvature. More often the ulcer occupies the posterior rather than the anterior gastric wall, but in the duodenum it is a little more common to see two ulcers, one anterior and one posterior, but here also in the great majority of cases there is but one. The average size is that of a dime (approximately 2 cm.), but it may be smaller and not infrequently is larger. The mucous membrane and submucous tissues alone may be involved, but in most chronic ulcers the destructive process has extended more deeply, into the muscular coat and even to the peritoneal covering of the stomach or duodenum. With such a variety of possibilities afforded as to situation, size and depth, it becomes evident what a great variability in symptoms may be produced by either gastric or duodenal ulcers; how uncertain is their course; how difficult it is to effect their healing; and how by obstruction of the stomach's outlet, or by erosion of vessels in its wall, or by complete penetration into the peritoneal cavity, an ulcer may produce the most serious and alarming consequences.

### CLINICAL HISTORY

The question as to whether there is a typical ulcer history, reliable enough to be accepted as a factor in diagnosis, has long

constituted a matter for controversy. To the writer it has always seemed that the story told by the patient with chronic ulcer is so characteristic that from this alone the diagnosis becomes probable, though never from this alone does it become certain. Experience teaches that now and then the apparently typical history may really be produced by some pathology entirely outside the stomach, or that no such history as expected may be elicited even when other methods of investigation and subsequent developments prove that the patient has a chronic ulcer in the stomach. Nevertheless, admitting that such exceptions to the rule occur, it still seems true that in the great majority of instances a careful history is to be depended upon as an important factor in diagnosis.

The features that make this history at least suggestive are chronicity, periodicity, rhythmic sequence of events and the character of the symptoms. By chronicity is meant the fact that the patient's disturbance of digestion has persisted for years, as many sometimes as five to twenty, and always at least for months. But this disturbance is not constant, and so periodicity stands out as the second peculiar feature. There are days, weeks or months during which discomfort or suffering persists, and then there are similar periods when the trouble disappears entirely or to a great degree. These alternating attacks and remissions vary in length, but they form a part of the story in practically every case of ulcer. A third remarkable point in the patient's account is the rhythmic sequence of events, while the attack is in progress. Usually the appetite is good and often it is keen, but about one to four hours after a meal symptoms of disturbed digestion appear. These persist and increase in intensity until the next meal, unless something is done meantime to relieve them. Taking food or an alkali

such as bicarbonate of soda again brings comfort for a variable period, and then the cycle repeats itself. This peculiar behavior of the stomach and the almost clocklike regularity with which the symptoms recur usually has been noted by the intelligent patient and constitute a part of the history. Finally the character of the symptoms in ulcer is sufficiently constant to make a distinctive clinical picture. About two hours after eating, sometimes earlier, sometimes later, discomfort is felt in the epigastrium, gradually increasing to more or less severe pain. Commonly this sensation is described as burning or as a dull ache, but it may be intense and lancinating pain. Belching of gas follows, and with this is apt to come a mouthful of hot, sour, irritating fluid that burns the throat and sets the teeth on edge. This belching and water-brash persist at intervals, and the stomach feels sour and upset. Sooner or later nausea forms a part of the distress. If this leads to vomiting and the stomach expels a quantity of sour, disagreeable contents, relief follows, but this may not be complete, until repeated vomiting has removed all the offending material. If the vomitus contains brown flecks or is tinged red by blood, this is due to a complication and should not be looked upon as a part of the routine ulcer history. Hemorrhage, even slight in amount, should never be a symptom essential to the diagnosis.

Such a history as described is the one usually elicited as regards chronicity and periodicity, but there may be considerable variation as regards the time relation of symptoms to meals, their character and their degree. The time of onset may be as early as one or as late as four hours after food is taken, or anywhere between, or shortly before the next meal is due, which leads to the designation "hunger pains". In some cases the worst suffering comes at night, rousing the patient from

sleep at midnight or later and disturbing rest for hours. The symptoms vary in character in different patients or in the same patient in different attacks. Sometimes there is but little pain experienced, and the complaint is more of sour stomach with heartburn, belching and water-brash. There may be no nausea or vomiting. But pain usually is felt at one time or another by the patient with ulcer, though it varies greatly in its intensity. Its variability in degree depends mainly upon the extent of the pathology, but the state of the patient's nervous system must always be taken into account as well, for some bear pain much better than others and make less complaint about it.

#### PHYSICAL EXAMINATION

The characteristic feature of this method of investigation in gastric ulcer is that it shows no objective sign of disease. Usually the only evidence obtained is subjective, that is, what the patient and not what the examiner feels. Tenderness at some spot in the epigastrium, the right hypochondrium or sometimes the left, is practically all that can be expected. But this sign depends so largely upon the patient's sensitiveness to pain that neither its presence nor its absence is conclusive. There is no objective sign of ulcer, nothing that can be seen or palpated. The finding of a mass should always suggest some other pathology, though a large callous ulcer sometimes can be distinctly felt through a thin abdominal wall. On the other hand negative evidence, the entire absence of any abnormality on physical examination except tenderness at some point in the upper abdomen, does not prove that the symptoms of gastric disturbance are due to ulcer, for no more than this may be found when chronic appendicitis or cholecystitis is respon-

sible for the patient's complaints. In general, therefore, the conclusion may be accepted that physical examination affords no positive proof that ulcer of the stomach is present, but that negative evidence neither proves nor disproves its presence.

### GASTRIC ANALYSIS

Of all the methods of investigation available for the diagnosis of gastric ulcer, none is more important than examination of the stomach's contents and secretion. What it contains in the early morning after a night's fast, and what it secretes in response to the stimulus of a test meal, give information of the greatest value as to whether ulcer is or is not present. But this evidence must always be considered in connection with that obtained by all other methods as well. By itself it should never be accepted as conclusive.

The fasting contents, withdrawn in the morning before the patient has taken food or drink of any kind, usually but not invariably present several characteristic features, when there is an ulcer in the stomach. The amount of material obtained frequently is increased above the maximum normal of 50 c.c., due mainly to interdigestive hypersecretion but partly to pylorospasm that favors accumulation. The total acidity and the free HCl are above the average normal and may even equal or exceed the values found in response to a test meal. There are gross fragments of food refuse, visible to the naked eye, or at least vegetable and muscle fibres found with the microscope in smears from the sediment after filtration. The whole material recovered may be blood-stained, or blood may be seen as streaks or brownish shreds, or it may be discovered only by the benzidin test and by red corpuscles seen under the micro-

scope in smears from the sediment. All of these findings, however, may be missing and the fasting contents practically normal, even when there is an ulcer in the stomach.

In the fractional analysis after a test meal hyperchlorhydria is the characteristic feature, the values gradually rising with each extraction so that the total acidity and the free HCl are decidedly above the normal throughout and higher at the end of an hour or even two than at any time preceding. But hyperchlorhydria is not essential to diagnosis, and ulcer may be present, even though fractional gastric analysis shows a normal secretory curve or even below the normal average. As a rule, the nearer the ulcer is to the pylorus, the greater the degree of acid in the secretion, while the farther away from the pylorus, the more likely it is that a normal secretion will be found. Even when hyperchlorhydria is discovered, it must be remembered that several extragastric diseases may reflexly produce this effect, and that this alone never justifies the inference that ulcer is responsible. The amount of secretion in response to a test meal, especially after histamine injection, usually is increased, when there is an ulcer in the stomach, as well as the amount of free HCl, but this is not invariably true. The combination of hypersecretion and hyperchlorhydria, however, is always a suggestive one.

#### STOOL EXAMINATION

Too much importance should not be ascribed to occult blood in the stool as a sign of ulcer of the stomach. All ulcers do not bleed, so that no visible or occult blood may be found even when ulcer is causing the symptoms. On the other hand, when blood is discovered, it does not follow that ulcer caused it to appear, because there are many other possible sites for its

origin between teeth and anus. The absence of blood from the stools does not prove that there is no ulcer in the stomach, and its presence there is significant only when every other possible source has been excluded. Occult blood in the stools is, after all, an indication of the activity of an ulcer rather than of its presence; once ulcer has been demonstrated, the continuance or disappearance of occult blood furnishes a guide as to the progress of healing. This is apparently the main value of this sign.

### X-RAY EXAMINATION

This method of investigation has become practically indispensable in the recognition of gastric ulcer. It gives demonstration, while all other methods so far described lead only to inference. The information it supplies concerns the size of the stomach, whether or not there is a defect in its contour and where this defect is situated, the presence of obstruction at the pylorus and its degree, the rate of peristalsis and the emptying time. Details by which these conclusions are reached belong to the x-ray technician, who obtains them by means of fluoroscopy as well as by films. But the clinician must measure the significance of the report rendered and must consider it in connection with all other evidence previously obtained by other methods. In the great majority of cases x-ray evidence is reliable and of the greatest importance, but it is not infallible. It is possible for an ulcer to be overlooked, or for one to be reported when it really does not exist; acceptance of x-ray findings to the exclusion of all others occasionally leads to error. It is equally true, however, that x-ray evidence is sometimes correct when all other evidence is against it.

## GASTROSCOPY

Those who have had experience with the gastroscope, and especially with the new flexible Wolf-Schindler instrument, report upon the safety as well as the value of its use in ulcer of the stomach. It shows the presence of such a lesion, its exact site, its size and the condition of the mucous membrane around it. Even in the presence of bleeding Jackson advises gastroscopy, in fact considers hematemesis a strong indication for its use. The gastroscope enables the bleeding area to be accurately localized and visualized in a way that can not be otherwise accomplished. With the straight tube it is also possible to secure a bit of tissue from an ulcer's edge for biopsy and thus for differential diagnosis from cancer. If this procedure is feasible in the œsophagus or a bronchus, it ought to be equally so in the stomach as well, but only when done by experienced hands.

## DIAGNOSIS

The pieces of evidence that finally combine to make complete the picture puzzle of gastric ulcer are the following: *First*, a history of long-standing digestive disturbance, lasting over months or years, with periods of exacerbation and remission, with characteristic features such as the onset of symptoms one to four hours after meals, with complaint of burning, belching, water-brash, sourness, more or less pain, nausea and vomiting, with relief obtained by vomiting, by soda, or by more food, with comfort persisting for a variable interval until the cycle begins again. *Second*, after thorough examination of the abdomen no sign of disease except tenderness over some spot in the epigastrium or either hypochondrium, but not

always even this. *Third*, abnormal amounts of free HCl found in the fasting stomach contents and after a test meal. *Fourth*, x-ray demonstration of a filling defect in the contour of the stomach. When all these symptoms and signs are assembled it is easy to recognize the picture they make. But unfortunately some of the pieces of the puzzle are sometimes missing, and when found, they do not always fit. The so-called ulcer history is largely a hyperacidity history, and the hyperacid secretion may be caused by something else besides ulcer. Physical examination that gives negative results is not limited to ulcer. Gastric analysis, when it shows hyperchlorhydria, does not show what causes it, and when it shows normal or even subnormal secretion, does not eliminate the possibility of ulcer. Finally, while x-ray examination usually makes the diagnosis certain by demonstrating the ulcer crater or filling defect, it does not invariably do so even when ulcer is present. It is the consideration of all the evidence taken together and never of any one part by itself that leads to a reliable conclusion.

Of all the problems that confront the diagnostician, when the patient's complaint is of a chronic painful stomach ailment, the most important one is the determination whether *cancer* is responsible rather than ulcer. The difficulty is made greater by the fact that what begins as ulcer may ultimately become cancer. Admitting the doubt that exists in many minds about the frequency with which cancer of the stomach develops on an ulcer base, there seems to be no question that this change does sometimes occur. There are certain differences, however, upon which dependance is placed at the time the patient seeks advice, in the effort to distinguish one form of disease from the other. There is usually a shorter duration of the history of

illness in cancer, which includes a few months or a year at most, with no periodicity, no remissions and exacerbations, but steady progress, and no such characteristic delay after meals before symptoms appear as in ulcer. On the other hand there is earlier loss of weight, color and strength in cancer and more obvious deterioration of general health. Gastric analysis usually shows deficient or absent secretion in cancer, but may not do so in the cases beginning as ulcer; even in recent cases with no previous ulcer history, secretion may be fairly well preserved for a long time. Finally, the x-ray shows a more ragged, irregular defect in cancer than in ulcer, and the extent of this usually is greater. In spite of all evidence, however, error now and then occurs, as the following case demonstrates.

A man, aged 49, complained that for a year past he had sour stomach requiring soda for relief, but no pain until about two months previous. This had persisted ever since, in spite of a diagnosis of ulcer and treatment by Sippy diet and alkaline powders. Physical examination was negative. Gastric analysis showed total acidity as high as 80 and free HCl 56. X-ray films demonstrated deformity of the gastric antrum, interpreted to mean chronic ulcer with inflammatory œdema about it. Increasing obstruction at the pylorus and vomiting at times of a frankly bloody fluid led to exploratory operation a few months later. At this time the total acidity after an alcohol meal was still 76 and the free HCl 58. Operation revealed a hard, cicatricial mass surrounding the pylorus. But even with this mass visible and palpable, it was thought by the surgeon to be chronic ulcer so that a gastroenterostomy was done with no preceding pylorotomy. For several months afterward he seemed greatly improved. Then all symptoms recurred with progressive loss of weight and color. A second operation, six months after the first, revealed a large carcinoma at the pyloric end of the stomach, fixed to the liver and too extensive to justify any attempt at removal.

It is a curious fact how often the stomach makes the chief complaint when the real pathology is elsewhere. Thus it happens that diseases of other organs than the stomach may closely simulate gastric ulcer. The explanation lies in the disturbance of secretion, and the hyperchlorhydria they induce reflexly. Among the group of extra gastric imitators of ulcer *chronic appendicitis* holds the first place. But after all the history of the two is rarely just the same. There may be more or less constant complaint of sour stomach, characterized by burning pain appearing at a variable interval after eating, belching of gas, sour eructations and even at times vomiting of sour fluid, and this indigestion may monopolize the patient's attention and predominate in the history. Nevertheless inquiry usually will bring out the additional facts that pain is felt at times in the right lower abdomen, attributed to gas, that there is disturbance of bowel function shown by chronic constipation replaced at times by diarrhoea for a few days, and that in some instances there have been intercurrent attacks of pain of greater severity in the right lower abdomen with tenderness and slight fever, lasting for a few days. Physical examination may be entirely negative as in ulcer except at the time of an exacerbation and then reveal only localized tenderness. Gastric analysis shows only hyperacidity, which may be as great in degree as that seen in ulcer or even greater. X-ray films may give no sign at all of abnormal conditions in or about the appendix, or they may call attention to such peculiar features as that it is kinked or adherent, or contains fecoliths that segment its outline, or that it empties very slowly after it is filled with barium. But whether fluoroscopy and x-ray films do or do not implicate the appendix, they at least fail to show the characteristic signs of gastric ulcer. In spite of every care

in weighing the value of evidence supplied by history, physical examination, gastric analysis and x-ray reports, it is not always easy to differentiate. Furthermore it has been repeatedly proved that both gastric ulcer and chronic appendicitis may be found in the same patient, which accounts for the occasional failure to distinguish one from the other before operation or autopsy.

A man, aged 42, had been troubled for about four years by pain in his stomach and nausea, but not constantly, although recently his attacks had lasted longer and his intermissions were more brief. His pain came on about two hours after eating and was relieved temporarily by soda. Once he had noted, about four months preceding his first visit, that his stools were as black as tar for two days. No evidence of organic disease was found in the abdomen on physical examination. Gastric analysis showed a secretion with acidity within normal limits. The x-ray examination showed the crater of a small ulcer in the middle third of the lesser curvature as well as a definitely deformed duodenal cap. The patient improved steadily on routine ulcer treatment, but a month after his first visit he had a sudden attack of pain in his lower abdomen with rigidity and tenderness and temperature rising to 102° F., lasting for several days. After that his symptoms all disappeared, he gained weight and felt well. But one year after his first acute attack of lower abdominal pain with fever he had another. This time it was thought best to operate at once. The appendix was found swollen, filled with pus, about ready to burst. After its removal, all digestive disturbances disappeared, the patient gradually gained twenty pounds in weight and has remained well ever since.

Next in frequency to chronic appendicitis or even exceeding it as an impersonator of ulcer of the stomach comes *chronic cholecystitis*. It is true that many patients with this form of gallbladder disease have the gastric secretion decreased so that hypoacidity or even complete achlorhydria is found. In a

smaller group, however, there is a reflex hyperchlorhydria, and then disturbances of gastric function may mimic those produced by gastric ulcer. Heartburn, belching, water-brash and more or less severe pain appear one hour or two after meals, relieved by soda, by vomiting or by taking more food. Physical examination usually is negative except possibly for epigastric tenderness. Gastric analysis shows hyperchlorhydria. Final decision as to diagnosis depends largely upon x-ray examination. This shows no such defect in stomach contour as characterizes ulcer, while by cholecystography the gallbladder may be found abnormal in size, shape, ability to concentrate and to empty. In the majority of cases it is possible to decide by careful study which organ is involved. But sometimes both are diseased, and the picture then becomes so confused that certainty in differentiation is impossible.

There are a number of other extragastric diseases that occasionally present symptoms resembling those of ulcer of the stomach and that may be mistaken for it. In these reflex hyperchlorhydria disturbs digestion, and the symptoms thus produced by the stomach attract the patient's attention more than those due to the original disease elsewhere. But if careful attention is paid to details in the history, if physical examination is made of every organ outside the abdomen as well as in it, if laboratory examinations include not only stomach contents and feces but blood, urine and possibly spinal fluid, and if x-ray films and fluoroscopy have been employed as aids, there should be no error in the conclusion ultimately reached. Failure to recognize the truth usually is the result of incomplete observation or of hasty judgment. This group of extragastric diseases includes those where the real pathology lies within the abdomen, as in the large bowel, or the rectum, or in bladder,

ureters or kidneys, or in the liver, or in the pelvic organs in women or the prostate in men. But rarely it may include diseases originating outside the abdomen entirely, but disturbing gastric function in a way that suggests ulcer.

Finally there remains that group of diseases designated *gastric neuroses*, where no structural change can be found either in the stomach, in other abdominal organs or anywhere else in the body to explain the patient's indigestion. In this group disturbance of gastric function not infrequently produces symptoms resembling those of gastric ulcer and distinguished from it only by careful elimination of all the features relied upon for its recognition, as well as by the failure to discover pathological change in appendix or gallbladder or large bowel or any other organ that might reflexly produce the hypersecretion upon which the symptoms depend. A diagnosis of gastric neurosis should never be accepted without thorough investigation to exclude organic disease, and even then constant watchfulness may ultimately detect developments that lead to a revision of this conclusion.

### TREATMENT

It is essential in the management of chronic gastric ulcer to recognize differences in the condition of various patients at the time they seek advice and to individualize the plan of treatment according to what is found after careful investigation. The great majority of patients with uncomplicated ulcer of the stomach respond satisfactorily to ambulatory measures, without confinement to bed, without loss of time from their occupation and without the expense of hospital detention. This statement is based upon a long experience with such cases throughout a number of years.

I. *Ambulatory Treatment.* — The routine plan employed for this group of patients has been found satisfactory in most instances, but exceptionally it does not suffice and some modification has to be substituted. It includes dietetic and medicinal details. The patient is given the following diet list at the outset:

7 A.M. Two soft-boiled or poached eggs; thoroughly toasted bread or zwieback or toasted soda crackers, with butter; a glass of milk, or a cup of cocoa made with milk and cream. 10 A.M. A glass of warm milk (about half a pint). 1 P.M. Beef, mutton or chicken, picked into shreds while raw or chopped fine, then made into a meat ball and cooked rare; toast, zwieback or crackers with butter; a glass of milk. 4 P.M. A glass of warm milk. 7 P.M. A bowl-full of well-cooked rice with butter or cream and sugar, or shredded wheat biscuit, toasted crisp, with butter or cream, or toasted bread or zwieback or crackers; with any one of these a glass of milk.

With this diet the patient is instructed to take one tablespoonful of olive oil just before each of the three main meals. In some instances, where it appears that pyloric spasm plays a part in the production of pain, for the olive oil before meals is substituted tincture of belladonna in dose of five to ten drops in one tablespoonful of water. One or two hours after the three main meals, depending upon the promptness with which symptoms appear after eating, one level teaspoonful of a powder is advised, consisting of bicarbonate of soda two parts, magnesium oxide one part and subcarbonate of bismuth one part. This can be taken stirred in a little water, but it does not readily dissolve. This powder acts not only as a sedative to the irritated stomach by neutralizing acidity, but also as a regulator of the bowels. If they are constipated, the dose may be increased to a rounded teaspoonful or more, or the more

laxative heavy oxide of magnesia may be substituted for the ordinary form. If on the other hand the bowels are inclined to move too freely, the dose should be reduced or a larger proportion of bismuth should be added to the prescription. In some patients also calcium carbonate seems to give more relief than bismuth subcarbonate and may be substituted for it in the prescription in the same proportion. If in any case discomfort recurs in the night, a glass of milk may be taken when the patient is awakened, followed later by the powder if needed for relief.

After a week or two on this plan the average case is sufficiently improved to justify allowing a more liberal diet. Then a second list is provided, from which selection may be made as desired, as follows:

*May Eat:* Eggs, soft-boiled, poached or scrambled lightly; tender beef, mutton or chicken, chewed thoroughly; sweetbreads or brains; any kind of fish, including oysters, cooked in any way except fried; cocoa made with milk and cream; milk as much as desired, with cream; no vegetables unless prepared as a purée except baked or mashed potatoes; soups of any kind, if not highly seasoned, cooked with rice or barley or vegetables, but strained clear after cooking; cream soups made from milk and the purées of vegetables; any cereal, provided that it is cooked for at least three hours, and the husks and coarse particles are removed by the purée sieve; white bread thoroughly toasted, soda crackers, zwieback, shredded wheat biscuits, served with butter or cream; cream cheese; cooked fruits, such as baked apple or pear, or apple sauce, or stewed dried fruits, as prunes or peaches, if put through a purée sieve; for dessert, rice pudding, corn starch, blanc mange, custards. *Avoid* coarse foods, such as hard-boiled or fried eggs; tough meats or meats cooked too long; or pork, veal or ham; vegetables, such as corn, string beans, peas or spinach, unless prepared as a purée; coarse cereals, such as oatmeal, or cracked wheat unless strained through a purée sieve; all hot breads and fresh

fruits; all irritating foods, such as very salty, sour, peppery or highly seasoned dishes of all kinds, including salt fish, pickles, salads, acid fruits and drinks, and highly seasoned soups; and all stimulating drinks, such as coffee, tea, and all alcoholic liquor.

With this second list the patient is instructed to continue the glass of milk at 10 A.M. and at 4 P.M., the olive oil or the belladonna before the three main meals and the alkaline powder one or two hours after them. This new plan is continued for from three to six months with the gradual addition of new articles of soft food, but cautiously and adhering mainly to the original list. All medication may be discontinued as symptoms disappear, but even after the patient feels perfectly well, care about diet must persist indefinitely.

During the past decade attention has been called by various observers to the possibility that alkalies in the treatment of ulcer given in too large doses or continued too long even in moderate doses may produce a condition of *alkalosis*. This occurs in only a small proportion of the cases where alkalies are used as a routine, probably in not over two or three per cent., but it may present a misleading clinical picture and should not be forgotten. A recent review of alkalosis complicating the treatment of peptic ulcer has been made by Jeghers and Lerner (New England Journal of Medicine, June 18, 1936, p. 1236). They note that the symptoms this produces resemble those of nephritis with nitrogen retention and possibly uræmia. The manifestations include nausea and vomiting, dizziness and headache and various mental disturbances. The urine is alkaline, increased in amount, with low specific gravity, often showing a trace of albumen and occasionally granular casts. The phenol-sulphophthalein test reveals a decreased excretion of the dye as low in some cases as 10 per cent. The blood chlorides are greatly

reduced, and the blood nitrogen is increased above 40 mgm. per cent. and may be even as high as 100 mgm. Treatment consists in discontinuing the alkaline powders entirely and administering sodium chloride by mouth, by rectum or by vein, at least twenty grams per day altogether by one or the other or all of these various routes, until the blood chlorides reach a normal level. Usually improvement rapidly follows such a plan..

II. If the patient's suffering seems unusually severe, or if no relief is afforded by ambulatory treatment on the plan described, then more vigorous measures must be adopted. In these cases the probable explanation for the greater severity and persistence of symptoms is that the ulcer is deeper and more extensive than usual. Now the plan of treatment recommended becomes practically the same as that proposed years ago by Sippy and known by his name. Rest in bed becomes essential. At the outset three ounces (90 c.c.) of a mixture of equal parts of milk and cream are given every hour from 7 A.M. to 7 P.M., with an alkaline powder, stirred in two or three ounces of water, midway between each feeding. Sippy powder number 1 contains ten grains (0.6 gm.) each of heavy calcined magnesia and sodium bicarbonate; powder number 2, ten grains (0.6 gm.) of calcium carbonate and twenty grains (1.3 gm.) of sodium bicarbonate, and these are given alternately between the feedings. This routine should be continued for at least two full days. On the third day, if all goes well, a soft egg with a cracker may be added to one of the forenoon feedings, and three ounces (about two large tablespoonfuls) of a well-cooked cereal such as rice, or farina or germea, may be added to one of the afternoon feedings. Gradually the egg and the cereal meals are increased in number

until at the end of the first week, two or three of each are given every day in addition to the regular three ounces of milk and cream mixture every hour, and at the same time as the latter. The powders are continued on the half hour.

In the second week it has been the writer's custom, deviating from the teaching of Sippy, to increase the amount of milk and cream mixture to four ounces (120 c.c.) and the interval between feedings to two hours. The powders are continued on the alternate hour. Other soft foods are also added now, such as custards, cream soups made from milk with vegetable purées added, and vegetable purées themselves without milk. These foods are permitted only at the time of the regular milk and cream feedings on the two hour plan.

In the third week it is usually possible to return to practically the first diet list and medication employed in the ambulatory treatment, but with such modifications as the condition of the patient makes advisable at the time. In this third week the patient may also be allowed to get out of bed for at least a part of each day. In the fourth week, after gradual trial of the patient's strength and digestive capacity, he may be allowed still more freedom as to activity and diet, and the second diet list will then become the one for permanent use subsequently for the following weeks or months. But no routine can ever be adhered to exactly as planned, and judgment must be used in deciding when and how to modify it.

Besides treatment by rest, diet and medication the patient with gastric ulcer must receive instruction about the importance of other matters relating to his disease. All foci of infection about the mouth, throat and sinuses must be removed, for they may keep up the activity of an ulcer or cause its relapse after it has once healed. Unusual physical or mental fatigue

should be avoided, because it tends to prevent the ulcer's cure. It has been proved that over-indulgence in tobacco retards recovery, so that smoking should be abandoned entirely or else greatly restricted. Alcoholic drinks likewise are harmful and ought to be excluded. Finally it should be made clear to the patient with ulcer of the stomach that in spite of any kind of treatment, relapse is common unless care continues to be taken after apparent cure. Particularly it should be impressed that the time never comes when beer, wine and other alcoholic drinks can be taken with impunity.

III. *New methods of treatment.* — A number of new ways to treat gastric ulcer have been proposed during the last few years. Some of these have received trial of sufficient extent by competent observers to make them worthy of use in selected cases. Others have been suggested and their claims advanced without enough experience as yet to justify their general employment. Foremost among these new methods of treatment is that by gastric mucin.

For five or six years past Fogelson has been trying to determine the efficacy of gastric mucin in the treatment of gastric and duodenal ulceration, and recently he has reported the results in approximately 555 cases observed during the previous three years. These patients were followed not only by Fogelson himself but also by a gastric mucin committee appointed by the Northwestern University Medical School of Chicago and by other physicians sufficiently interested to co-operate by reporting, on a questionnaire furnished them, the data at outset and during the progress of treatment.

Detailed statements were given by these different men, to whom mucin was supplied for use in treatment, after the material had been accepted by the committee as suitable and proper

in every respect. These statements covered the history of the case, the x-ray findings, recurrences, incidence of hemorrhage and the effects of former treatment, including surgical procedures as well as the progress of the case and the effects of treatment by mucin. The average dose was 80 to 100 grams of dry mucin powder, suspended in a mixture of milk and cream, at frequent regular intervals, as often as every hour in acute cases. The diet was limited to the bland substances usually prescribed in ulcer. The treatment was continued for at least six months, the dosage, however, being reduced during the later stages. The results were highly satisfactory, considering the fact that only cases were selected for treatment that were previously considered intractable, because they failed to respond to ordinary methods. Of 555 who took mucin from six months to three years, 348 were rendered free from symptoms, 114 were partially improved and 93 failed to secure relief. Of the latter, however, only 32 were counted true failures, because the other 61 for various reasons would not continue the use of mucin for an adequate period. Recurrence of symptoms while still receiving mucin was noted in 6 persons, acute massive hemorrhage in 4 and perforation in one. In 56 patients with a recurrence of symptoms after gastroenterostomy, suggesting a new ulcer at the gastroenterostomy stoma, complete control of all the subjective symptoms was obtained by the use of mucin in 36, partial relief in 16 and no relief in 4. The one important question not yet answered is the permanence of the results obtained, and this can not be answered until more time has elapsed. Certainly the method is worthy of trial, particularly where other plans of treatment have failed.

The only other one of the newer methods of treatment that seems to deserve more than passing mention is that by histidine

monohydrochloride, which has been extensively advertised to the medical profession under the trade name larostidin. This remedy is to be given by parenteral injection, daily or every other day, until twenty-four ampoules of 5 c.c. each are administered. Reports on the use of this remedy have not been altogether satisfactory. Among the most recent is that by Sandweiss of Detroit (*Jour. A. M. A.*, April 25, 1936, p. 1452), who treated 40 patients by this method. The objections he finds to it are its cost, the fact that twenty-four visits by the patient to the office or clinic are required, that in an appreciable number of cases mild reactions are produced, such as weakness, weariness and various aches and pains, that there is a high percentage of recurrence of symptoms within six months after treatment, and that approximately the same percentage of patients respond favorably to the diet-alkali regime without histidine. It produced remission of symptoms in 55 per cent. of the group treated by Sandweiss, but did not prolong the symptom-free interval nor prevent recurrence, and 85 per cent. of patients, who developed remissions, returned with ulcer symptoms within six months after treatment. The remedy may be used in cases not responding to the dietetic, alkali, anti-spasmodic routine commonly employed, but only for temporary relief, not for cure, because in 24 patients checked by x-ray or by operation after finishing the course of histidine, Sandweiss found that not one showed disappearance of the ulcer deformity. Almost identical conclusions are reached by Martin of New York (*Jour. A. M. A.* April 25, 1936, p. 1468). Of 41 cases treated by histidine 30 were symptom free at the end of four weeks, but 24 of these 30 had one or more relapses within ten months. It is significant that the Council on Pharmacy and Chemistry of the American Medical Association has found larostidin not acceptable for in-

clusion in the volume of New and Non-official Remedies because it is marketed with unwarranted therapeutic claims.

Every medical journal in recent months contains advertisements of a variety of new remedies and methods of treatment for ulcer of the stomach with arguments in favor of their adoption, but the wise physician will await the judgment of time before substituting any of these for the older and well-tried measures that have won the confidence of the profession.

CHAPTER V

COMPLICATIONS OF ULCER OF THE STOMACH

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While gastric ulcer ordinarily causes more or less constant discomfort or even at times severe pain with incapacity for concentration on work, it does not threaten life until complications develop. These are always possible, however, in every case and may present themselves at any time unless measures are taken to induce healing of the ulcer, or even in spite of such treatment or in its course. These complications include (1) pyloric obstruction, (2) hæmorrhage, (3) perforation and (4) hour-glass contraction.

PYLORIC OBSTRUCTION

Pyloric obstruction quickly leads to food retention, at first small in amount but gradually increasing until ultimately it may become very large. It is important to remember that such obstruction frequently is due to other factors than actual constriction by an ulcer scar. Inflammation about the ulcer, swelling and œdema likewise play a part, and pyloric spasm always is a possible additional obstacle to the passage of stomach contents onward. This understanding of the reasons

for obstruction is essential for proper treatment. The patient's symptoms, that suggest this complication has developed, are increased pain and vomiting. The attempt on the part of the stomach wall to overcome obstruction and force food through the pylorus in spite of it, means increased peristalsis, and these more vigorous waves thrown against the pyloric resistance result in increased pain. Furthermore, this effort at emptying goes on practically all the time, because the stomach is never free of its contents, and so the usual rhythm of pain only at the height of digestion is changed to pain more or less constantly. As the amount of residue increases, vomiting becomes the stomach's last resort. More and more material is gradually removed by this method, and the character of the vomitus is changed from the usual sour, irritating fluid of hyperacidity to that of fermented, decomposed food, with so-called "swill-barrel" odor. Or if vomiting is ineffective, accumulation of food continues until the patient in desperation takes decreased amounts or refuses it practically altogether, because of the distress it causes. Thus gradual loss of weight and strength follow from self-imposed starvation.

On physical examination, certain signs are discovered that are diagnostic. These are first the outline of the dilated stomach seen distinctly through the abdominal wall, and second a visible peristaltic wave across it, moving from left to right. If such a wave does not appear at once, usually it can be elicited by tapping the abdomen over the dilated area with the fingers or striking it lightly with the end of a towel. Or if this device does not succeed, the peristaltic wave almost certainly can be made visible by inflating the stomach with carbonic acid gas. This is easily accomplished by administering separately one teaspoonful of soda bicarbonate in about two ounces of water

and tartaric acid one teaspoonful in the same amount of water. The distension this causes not only demonstrates the enlarged contour of the dilated stomach but also induces vigorous peristaltic waves. Thus the fact is proven that pyloric obstruction exists, even though no information is given about the cause.

When the stomach tube is passed to obtain fasting contents for analysis, a quantity of material is found manifestly retained at least over night. Sometimes the amount of refuse found in such stomachs is astonishing, and repeated lavage is required to remove it and to make the stomach clean. A test meal is out of the question until this is done, because the extraction for analysis gives results of no value. Even after the stomach is emptied, the analysis usually shows a secretion below normal, because long retention has impaired its secretory power by the chronic gastritis it has induced.

X-ray films demonstrate the pyloric obstruction by the retention of barium for hours after it has been swallowed, but it is more difficult to discover by means of the films or fluoroscope examination what sort of disease has caused the obstruction. Sometimes a clear cut crater is shown at the pylorus or near it that speaks definitely for ulcer. Sometimes the defect visualized is ragged and irregular, such as cancer produces. Not infrequently the pyloric end of the stomach is blunt and rounded and shows no characteristic lesion of any kind. It is not possible, as a rule, to decide from x-ray films alone without other evidence what has caused the interference with the pyloric outlet. Previous history of the patient, before this complication arose, helps greatly in solving the problem.

A man, aged 42, complained that for two or three years he had suffered at intervals from pain after eating, usually appearing soon

after a meal but lasting for hours, with nausea and occasional vomiting. He was frequently awakened by pain about midnight, relieved by soda. His abdomen showed a very definite peristaltic wave across the stomach from left to right but no palpable mass. Gastric analysis gave a secretion within normal limits. X-ray films revealed a narrow and fixed pylorus with so great delay in emptying that not only at six hours but also at twenty-four it still contained practically all the barium swallowed. After lavage, with rest and ulcer diet, belladonna and alkaline powders, he was soon rid of all his symptoms and in three months was dismissed from observation. He remained well for seven years, then returned once more complaining of abdominal pain, vomiting and unwillingness to eat because it hurt him to take food. When lavage was done, a large quantity of sour, foul-smelling material was evacuated. Again by rest, Sippy diet and alkaline medication he rapidly recovered and in two months was back at his work, free from all symptoms. There has been no recurrence since.

The foregoing case illustrates that the diagnosis of pyloric obstruction does not mean that there must be immediate resort to surgery. In many cases acute inflammation and pyloric spasm are larger factors than actual organic blocking by cicatricial fibrous tissue. It is always justifiable, therefore, to make an attempt first to overcome the obstruction by non-operative measures. The plan that gives the best results in such cases is rest in bed for a few days or a week, washing out the stomach at least once at the outset, to remove retained and decomposed food, using for lavage a warm solution of bicarbonate of soda, one teaspoonful to a pint. This may have to be repeated every morning for several days. The diet should be restricted to the Sippy mixture of equal parts milk and cream, three ounces (90 c.c.) every hour from 7 A.M. to 7 P.M. An alkaline powder consisting of ten grains (0.6 gm.) each of heavy oxide of magnesia and bicarbonate of soda should be given on

the half hour between feedings, alternating with a second powder of calcium carbonate ten grains (0.6 gm.) and sodium bicarbonate twenty grains (1.3 gm.). Gradually soft-boiled egg and well-cooked cereal should be added, the milk and cream mixture should be increased in amount, and the interval between feedings lengthened, as described in the chapter on the treatment of uncomplicated ulcer. Sooner or later by this plan it is possible to resume the regular lists advised for use by ulcer patients.

If such dietetic and medicinal treatment fails to give relief, or if there are frequent recurrences after apparent cure, then surgery becomes necessary. Gastroenterostomy is the procedure usually indicated for pyloric obstruction, and this operation is successful in most cases but not in all. Why it fails in some instances is a question for surgeons to answer, but it seems to be admitted that failure usually means faulty technique. The following case affords an instance of an ideal result.

A woman, aged 35, who first sought the writer's advice in 1908, had been afflicted by stomach disturbance for the previous two years. Her story was quite typical of chronic gastric ulcer. A year before she had been found by her local physician to have an enormously dilated stomach with persistent food retention. She improved then by the use of daily morning lavage and remained comparatively well until three months before coming to San Francisco for advice. Then pain, belching, vomiting and weight loss recurred and persisted in spite of washing out the stomach every morning. Her stomach was found to contain a large quantity of foul-smelling refuse and required repeated lavage to make it clean. After efforts at relief by diet and medication had been carried out for a month without success, gastroenterostomy was advised. Following operation she recovered rapidly. This patient was last seen in 1935, twenty-seven years after gastroenterostomy, and reported that her digestion had never annoyed her since, unless she grew careless about the amount she ate at a meal.

## HÆMORRHAGE FROM ULCER OF THE STOMACH

Bleeding is not a symptom in every case of ulcer of the stomach, and its occurrence should always be looked upon as a complication. Statistics show it is observed in only 10 to 25 per cent. of all cases. Nevertheless it is always a serious incident in the ulcer's course. There is rarely any difficulty about recognizing the fact that blood expelled from the mouth comes from the stomach, though much doubt may exist as to whether ulcer caused it. The patient observes and reports that the vomitus is colored red or reddish brown or contains brown flecks, or that there is really bright red blood of varying amount in the vomited material. Such attacks, if slight, usually are brief in duration but are apt to repeat themselves. If they are not heeded and proper measures taken to stop them, sooner or later a large hæmorrhage is likely to occur. Such a large hæmorrhage may, however, be the first one observed, though this is unusual when ulcer is the cause. When it does appear, it is always a terrifying event. The patient seeks advice then, even though previous dyspepsia, sour stomach, pain and even an occasional brown tinge in the vomitus may all have been disregarded. The story of what happened is confirmed by the exhibit of clothing or bedclothes soiled by blood or of vessels containing the vomited material. The patient's pallor, look of anxiety, sweating skin and rapid pulse combine to prove the amount and rapidity of the blood loss, even though no blood is actually seen by the physician. Sooner or later the stools also show the presence of blood, in black tar-like masses, if much is present, or by chemical tests for occult blood, if the amount is small, for more or less always passes out of the stomach into the bowel besides that vomited. In such an emer-

gency the best plan is to omit palpation and percussion of the abdomen for the time being, because vigorous manipulation may start fresh bleeding. Gastric analysis likewise should be postponed to some future time. X-ray examination, involving the swallowing of barium for fluoroscopy or films, is to be avoided for days after hæmatemesis. There will be time enough later on, if the patient survives, to determine the source of the hæmorrhage. The immediate indication is to save life, and certainty about the diagnosis must wait.

The probabilities, however, with any gastric hæmorrhage are always in favor of ulcer, for it is the most common cause of hæmatemesis. A previous history of chronic dyspepsia and sour stomach and perhaps of recurrent small hæmorrhages preceding a larger one is characteristic of ulcer but can not always be obtained. At the moment all the physician can do is to recall other possible causes for hæmatemesis and the features each presents. Next to ulcer in frequency as a source of this catastrophe comes cirrhosis of the liver. But in this disease hæmatemesis more often appears out of a clear sky with no antecedent history of digestive disturbances like those in ulcer and with no preceding smaller hæmorrhages. In splenic anæmia, so-called for want of a better name, a large hæmorrhage from the stomach is a common manifestation, but the disease is rare, and the spleen is found enlarged as a characteristic part of the clinical picture. Cancer of the stomach seldom produces a large single hæmorrhage and practically never without a long period of severe indigestion going before, during which time there may be repeated small oozings of blood from the neoplasm and the vomiting of small, dark flakes of partially digested blood known as "coffee-ground" material.

In general there are a few well-recognized rules for guidance

in deciding what hæmatemesis means. (1) When in doubt, assume ulcer as the cause until some other has been proven. (2) An ulcer history usually precedes but the ulcer may be latent until hæmatemesis appears suddenly. (3) The quantity of blood vomited is variable, and the amount does not always indicate properly the seriousness of the situation. (4) One small hæmorrhage frequently is followed by others with a day or two interval between, and while the bleeding may start as a simple oozing with the discharge of a small amount of blood, it is apt to increase gradually if no precautions are taken to prevent. The common course is for small repeated hæmorrhages to occur at short intervals before there is a large one, but the first may be large enough to cause prostration and sometimes, though rarely, to prove fatal. It is imperative, therefore, to regard as serious every hæmatemesis, however apparently insignificant, and to act promptly in caring for it.

### *Treatment of Hæmorrhage from Ulcer*

Numerous plans of treatment for hæmorrhage as a complication of ulcer of the stomach have been proposed by different men, but there are certain particulars in its management upon which all seem to agree, and these will be considered first. No matter how slight the blood loss and of how little importance its effects seem to be, the first essential is *rest* not only for the stomach but for the entire body. This means that the patient should remain in bed with no unnecessary change of position, no rising for any purpose from the recumbent posture, no visitors at the bedside. These instructions are always better carried out in hospital than in the home, and there also more careful observation is obtained, such as trained assistants give. But the hospital is not essential in cases of small bleeding, and after

a large hæmorrhage it may not seem wise at once to move the patient. Every case must be judged for itself in this matter. Mental as well as physical rest is made more certain by the administration of morphine and atropine hypodermatically, one-quarter grain (15 mgm.) of the former with one hundred and fiftieth (0.4 mgm.) of the latter, repeated every four to six hours, as required to keep the patient quiet and partly drowsy. Nothing whatever should be given by mouth, no food, no water, no drugs. Ice should be applied continuously to the abdomen by the ice-coil or the ice-bag.

So far there will be little difference of opinion about the proper methods to employ, but beyond this point beliefs vary about what should be done. The writer has always felt convinced and has taught that it was better to avoid attempts to restore lost blood volume as a routine by the immediate use of normal salt solutions by subcutaneous infusion, or of glucose solutions by intravenous injections, or even of whole blood transfusion, for all these procedures interfere with the collapse of vessel walls and the formation of a clot that causes bleeding to cease. There is wide difference of opinion about this matter, however, especially as regards transfusion, and if the hæmorrhage has been so large as to cause extreme weakness and prostration, as shown by heart action and blood pressure as well as by the patient's appearance, transfusion should be done as soon as a proper donor can be obtained, even though theoretically it seems contraindicated for the reasons given. Transfusion, after all, is a more logical stimulant than strychnine, camphor, caffeine or digitalis, even though the pulse is weak and rapid, for these drugs all work at cross purposes with the general principle of rest. Weakness for the time being affords the patient the best defence against recurrence of bleeding.

Years ago, prompted by the recommendation of Ewald, the writer first used in the treatment of hæmorrhage from ulcer lavage of the stomach with ice-water, introduced repeatedly in small quantity until all clots were removed and the water returned clear. This plan has been adopted occasionally since, when the patient was seen early, and always with good results. In recent years it has been endorsed by Sippy and also by Hurst. The latter says in his monograph on "Gastric and Duodenal Ulcer" (page 270); "When the hæmorrhage is severe and continues after hæmatemesis has occurred, or when in spite of its severity it is not sufficiently rapid to cause sudden distension which results in its ejection by mouth, death may result unless steps are taken to empty the stomach. It is common knowledge that uterine hæmorrhage can almost always be arrested by emptying the uterus and causing it to contract, but it is less widely known that similar treatment for very severe gastric hæmorrhage is almost equally effective." Such treatment may seem heroic and dangerous, but in a situation as fraught with peril as the one for which the treatment is adopted, heroic measures are sometimes indicated. Furthermore, if the stomach tube is not inserted deeply, and if not more than four ounces (120 c.c.) of ice-water are poured in at one time, there is very little danger of doing harm.

The question how long to keep the patient's stomach empty after a hæmorrhage is another one about which opinions differ. Some authorities advise complete abstinence for as long as forty-eight hours. Sippy and Hurst both recommend this delay about resuming feeding. But as the hours pass, thirst becomes the most troublesome symptom, and the desire for water is more and more urgent. After the lapse of twelve hours, if no further hæmatemesis has occurred, it is usually safe to allow

ice-water by mouth, a half ounce (15 c.c.) every hour. Even this amount stimulates some degree of peristalsis and endangers dislodgment of a clot already formed, but ice-water is less likely to do this than any food. The amount allowed may be doubled, if twenty-four hours pass without further hæmatemesis. But it is undoubtedly safer to withhold food of any kind for forty-eight hours following the first evidence of bleeding. If no further sign has appeared by that time, it is justifiable to begin the three ounces (90 c.c.) of milk and cream mixture every hour, with the usual alkaline powders half-way between, gradually increasing the amount and adding other soft foods as already described.

If after feeding is resumed, another hæmorrhage occurs, judge of the seriousness of this by the amount of blood vomited and passed by bowel, by the heart rate and blood pressure and by a blood count to determine how far the hæmoglobin and red cells have fallen below the normal. Transfusion should be done now, if the patient's general condition seems alarming, but feeding and powders must be discontinued temporarily, and surgery must receive consideration.

When to abandon medical treatment for hæmorrhage in gastric ulcer and turn instead to surgical is a most difficult decision to make. Great as is the danger from repeated blood loss, uncertain as the outcome seems, it is necessary to bear in mind also the risks of surgical interference at such a time. Statistics show that only about one to two per cent. of gastric ulcer patients die from hæmorrhage treated on a medical plan. On the other hand surgeons themselves quite unanimously agree about the risk of immediate operation, and as Balfour states it: "the danger of succumbing to hæmorrhage is less than the danger of operation during hæmorrhage". Certainly

no hasty decision should ever be reached in these cases, prompted by the excitement of the patient's family and friends, who so often are terrified and begging for immediate action. Under such circumstances, it requires all a physician's courage sometimes to stand fast in his adherence to that which he thinks best.

A man, aged 38, seen first in 1901, complained of pain in his stomach after food, always worse at night, present no matter what kind of food was taken, with vomiting quite regularly since the pain began and always giving relief until food was taken again. The vomitus was liquid, sour and irritating but never contained blood. Physical examination was negative except for epigastric tenderness. After the Ewald test meal, the total acidity was 79, consisting mostly of free HCl. There were no x-ray films for diagnosis in those days. Furthermore there was no Sippy diet plan for treatment. But by rest in hospital and a diet of milk and lime water followed by soft foods, he made a perfect symptomatic recovery and remained well for a year. Then the symptoms all recurred. The Ewald meal now showed a total acidity of 100 with free HCl 70. This time the patient refused to go to bed and rest. He was treated as before by milk diet and alkalies, while he kept about at his work. There was no relief afforded, however, and the symptoms all persisted. After this attack had lasted a month, he had one evening, while lying quietly at home in bed, a profuse hæmatemesis, sufficient to make him lose consciousness. Then he was willing to go again to the hospital. Under treatment by rest, an ice-bag over the abdomen and rectal feeding the hæmorrhage did not at once recur. The technique of transfusion had not in those days reached its present perfection, so none was given. Ten days later, while still at rest, there was another profuse hæmatemesis. In spite of all treatment the bleeding continued daily thereafter, until finally, after another large hæmorrhage, the patient died. Autopsy showed an oval ulcer 2 cm. by 3 cm., lying across the pyloric ring, with a large branch of the gastroduodenal artery open in its

floor. In retrospect it seems likely that nothing except excision of the entire ulcer could have prevented this fatality.

In sharp contrast to this unfortunate outcome stands the history of this man's son, who sought advice in 1920.

This man, aged 31, complained he had had a sudden attack of hæmatemesis, which caused so much blood loss that he fainted. For several days after that he continued to pass blood by his bowel, but this gradually ceased. Before this experience he had been ailing for about three years, intermittently, with typical symptoms of ulcer, but had consulted no physician. Physical examination was negative. Gastric analysis was omitted because of recent hæmorrhage. X-ray examination showed a constant defect in the duodenal cap with slight delay in emptying time. Recalling what happened in his father's case, this patient was sent to a hospital, given a transfusion and referred to a surgeon. Again the ulcer found at operation extended across the pylorus, partly within and partly without the stomach. A gastroenterostomy was done, and no further trouble has occurred since.

#### PERFORATION OF ULCER OF THE STOMACH

By this is meant destruction of the stomach wall by the ulcer to such a depth that all restraint is overcome, and the stomach's contents are permitted to escape into the peritoneal cavity. This is but the consummation of a threat always present from the time that gastric ulcer is first diagnosed, but a threat rarely fulfilled because medical care and Nature's methods of management all tend to prevent it. It is a rare complication compared with hemorrhage, but even now, when early operation saves so many lives, statistics show that it remains the most common cause of death from ulcer. The physician's treatment to bring about healing is supplemented by Nature's efforts to avoid the catastrophe of perforation, for

as soon as an ulcer has extended deeply enough through the wall of the stomach to involve the peritoneal coat, a localized peritonitis results. By this process, which is protective, a patch of inflammatory exudate is placed over the spot where perforation is threatened. As a consequence the clinical history is modified, the pain that follows becoming more constant, more independent of digestion and more severe. If the peritonitis causes adhesions between the stomach and some organ behind it, such as the pancreas, there is the added feature of pain in the back, so that complaint is made of a boring, lancinating pain through and through. If the ulcer is on the anterior wall, so that there is no organ to afford it protection by adhesions, not only is perforation more likely to occur, but there is less of the antecedent severe pain that suggests perforation is threatening.

Besides the change in clinical history to warn the patient of possible perforation, even more definite proof of this danger is furnished by x-ray films, which show an out-pocketing defect or teat or projection beyond the contour line of the stomach wall. This, however, will not be produced if the stomach is attached to some other organ which the ulcer has made its base. Many such defects, however, are observed in ulcers which never perforate into the abdominal cavity or cause diffuse peritonitis. Then the changed character of the clinical history, greater local tenderness and possible increase in the amount of free HCl found in stomach contents because of greater nervous irritability, supply the only evidence to make the physician suspect that the ulcer has penetrated deeply, and that perforation is threatened. Only too often such evidence is lacking or is too indefinite to warrant interference by surgery, so that the final break in the stomach wall comes when least expected.

If all the barriers that Nature sets up are finally overcome, and the stomach wall is broken through, sudden agonizing, excruciating pain marks the entrance of stomach contents into the peritoneal cavity. The pain is felt at first in the epigastrium or right hypochondrium but later on becomes more diffuse. With the pain or shortly following it come nausea, weakness, faintness and a feeling of complete collapse. Physical examination shows the patient's face pale and covered with perspiration, the pulse rapid, weak and thready and the temperature at the outset normal or subnormal. As hours go by, peritonitis supervenes, the abdomen becomes exquisitely tender over the area where pain is felt, a protective muscle spasm makes the abdominal wall board-like in its rigidity, and the temperature gradually rises.

All of these peculiar features combine to make the diagnosis reasonably certain, particularly if there is a history of chronic digestive disturbance for months or for years preceding this sudden emergency. Occasionally the story may include the statement that the character of the long-standing symptoms has changed recently, and the pain has become more severe and constant, but this is not always the case. Possibly also a recent x-ray film of the stomach may have shown an out-pocketing defect to suggest the nature of the patient's present attack. The onset of perforation frequently is during a meal of greater latitude than previous directions had allowed. A patient recently observed had his attack during his dinner on Christmas day. In another case the catastrophe may follow some unusual physical effort, as in golf, tennis or bowling. But even so the previous history is not always characteristic enough to identify whatever disease preceded perforation. Thus it happens that a ruptured appendix or gallbladder may

be suspected instead of a gastric ulcer, and it is not always easy to decide which organ is responsible. Free air or gas in abdominal cavity, easily determined by x-ray, makes very probable the diagnosis that an ulcer has perforated. But it is usually possible to recognize promptly that perforation has occurred, and the cause is not of great importance. Immediate surgery is indicated in any event. Statistics show that the greater the interval between the accident and its repair, the smaller the chance for recovery. There is no justification for delay, once the diagnosis is made, and even though the patient's condition should prove to be ruptured gallbladder or appendix instead of perforated gastric ulcer, no harm is done by the error, except to the physician's pride.

A patient, aged 32, complained of pain and vomiting after meals more or less constantly for two years, but without hematemesis at any time. After a preliminary history and physical examination he was told to report on another day for gastric analysis. But after breakfast the next morning, while leaning over to wash his face, he was seized suddenly by very violent pain in his abdomen. When seen shortly afterward at his home, he was found in a condition of shock, very pale, prostrated, perspiring profusely, with a rapid thready pulse and subnormal temperature. He was taken as soon as possible to a hospital, a surgeon was called, and the abdomen opened within a few hours after the accident occurred. An ulcer was found on the anterior wall of the stomach, approximately two inches from the pylorus. Through this there was a perforation about a quarter inch in diameter, leading directly from the interior of the stomach to the peritoneal cavity. This opening had permitted food to escape which was found lying free outside the stomach. All foreign material was removed, the peritoneum cleansed, and the perforation closed by gastroplication. Three years later, when last seen, this patient reported he had no further trouble of any kind, weighed more than ever before in his life and was working hard every day.

The question is frequently argued, whether the patient with a distinct out-pocketing defect in the stomach shown by x-ray films should have excision of the ulcer crater at once because of the danger of perforation. Some authorities advise this course as a routine, but the writer has observed several such cases where by rest in bed, careful dieting and alkaline powders, not only have pain and all other symptoms ultimately disappeared but also the out-pocketing defect as well. (The editor believes that this is the rule, and his experience has been that most perforations occur when there has been no previous evidence by x-ray of any out-pocketing by the ulcer.) It seems safer, therefore, to try Sippy diet and the usual medication first and to reserve surgery for those patients who do not respond after a reasonable lapse of time.

#### HOURLASS STOMACH

This name is given to a deformity of the stomach characterized by a shape like that of an hour-glass with a pouch at each end and a constriction between the two. Almost invariably it is produced by a large ulcer on the lesser curvature that in the course of years has produced cicatricial contraction sufficient to divide the stomach into two compartments, with a passage-way still pervious between them, though of much diminished calibre. Such a lesion, extending downward from the lesser curvature to a variable degree over the anterior and posterior walls, has long been known as a "saddle ulcer". It is not a common complication, but neither is it rare. For some unknown reason it is found much more often in women than in men and usually in middle life or later, after years of ulcer history have preceded.

Symptoms after this complication has occurred are not dif-

ferent in character from those before, though they are apt to become more severe. Pain is the principal complaint, beginning as is the rule in uncomplicated ulcer, an hour or two after meals, or as late as three or four, but in this complication it is likely to become more constant and less intermittent. Vomiting takes place more frequently after this complication has developed, and large quantities may be evacuated, if there is obstruction at the site of constriction with retention in the upper compartment. Weight loss is then the rule, gradual and progressive, just as so often is seen in pyloric obstruction. On physical examination, there is found tenderness, more or less extreme, in the epigastrium or one or the other hypochondrium and not infrequently a palpable tumor, especially when the abdominal wall is thin. In such patients also a peristaltic wave sometimes may be observed running across the stomach from left to right just as in pyloric obstruction. Gastric analysis gives a variable secretion, and it may be low in free HCl, because the material obtained comes entirely from the upper compartment. But there is no evidence that makes the diagnosis of this complication certain, except x-ray examination, by fluoroscope and films. This will demonstrate the peculiar configuration of the stomach, if care is taken, though at first it may show only the upper pouch filled with barium. Time is required, if stenosis exists at the site of constriction, to permit the opaque material to pass through into the lower pouch. Previous to the addition of the x-ray to the methods available for the investigation of gastric disease, the diagnosis of this complication was seldom made except by exploratory operation, and this was done usually for pyloric obstruction and not for recognized hour-glass stomach.

The only other organic diseases besides chronic gastric ulcer

that may produce a similar deformity of the stomach are cancer and syphilis. As regards syphilis of the stomach, while theoretically it must always be considered a possibility, practically it is so rarely seen that the chance it is the etiological factor becomes almost negligible. As regards cancer, it likewise causes this deformity much more rarely than does ulcer. The story of more or less constant pain without the usual rhythm relative to meals, the finding of a tender palpable tumor in the upper abdomen in a patient who has gradually become emaciated and anæmic, all combine to suggest malignancy. But cancer gives no previous long-standing history of gastric disturbance, which may run back for years before hour-glass deformity develops. Yet on the other hand, the large lesser curvature ulcer with such an extensive previous history is the one that occasionally undergoes malignant degeneration, so that the time element must not be assigned too much value in differential diagnosis. X-ray examination can not tell positively that the ulcer has become cancer, but its size as demonstrated in the films has been claimed by some authorities to be a diagnostic feature. If an ulcer is shown to be one inch or more in diameter, it is said to be probably malignant and ought to be excised. But other authorities deny that this is always a reliable sign, and certainly clinical results fail to prove it true in all cases.

In deciding this very important question, when the duration of digestive disturbance is uncertain or seems suspiciously brief, when the character of the symptoms has not entirely corresponded to what is expected in chronic gastric ulcer and recent exacerbation has been accompanied by progressive loss of weight and strength, when a tumor is palpable in the upper abdomen, tender and fixed, and a peristaltic wave is visible

across the stomach, when the gastric secretion after a test meal shows a decreased or entirely absent free HCl, and when the x-ray examination reveals an hour-glass stomach with a large saddle ulcer on the lesser curvature, what conclusion shall be reached? Even in the presence of all this evidence it must be kept in mind how much more frequently chronic ulcer causes the gastric deformity in question than cancer ever does and how much greater are the chances in favor of the former than of the latter explanation, and this knowledge as a rule justifies delay and the trial of medical treatment before subjecting the patient to an extensive resection of the stomach wall, which is the only other alternative.

Another argument in favor of conservatism in dealing with such cases is the fact that hour-glass constriction of the stomach may be entirely spasmodic and not at all the consequence of cicatricial changes causing stenosis. This spasm may result not only when the cause is chronic ulcer on the lesser curvature but also when the ulcer is situated elsewhere in the stomach or even beyond the pylorus, in the duodenum, or even when there is no organic disease at all in the stomach but only in some other organ, such as the gallbladder or appendix. Such reflex spasm and the hour-glass deformity it produces occasionally will yield to belladonna and disappear even without cure of the disease that caused it.

If the patient with hour-glass stomach is treated exactly as if this complication did not exist and only as indicated by the large ulcer on the lesser curvature, all the symptoms usually subside, comfort is restored and lost weight is gradually regained. Rest, Sippy diet, alkaline powders, possibly belladonna, frequently accomplish all these purposes, so that the patient goes on in comparative health even though the hour-glass

stomach persists. If such a plan of medical treatment does not succeed in affording relief after a month or two of trial, it will then be in order to consider the advisability of surgery. The following case illustrates the possibility of managing this complication by simple means.

A woman, aged 73, complained that she had been troubled by her stomach at intervals for years past, "just indigestion," relieved always by taking soda. But for two months preceding her consultation, she had lost appetite, had almost constant pain increased by taking food, with nausea but no vomiting. Because of this she had eaten little and had lost much weight. Physical examination showed her weight to be only 102 lbs., and she was emaciated and anæmic. In the mid-abdomen was visible a protrusion, apparently the stomach distended by gas, with great tenderness in the epigastrium but no palpable tumor mass. Gastric analysis showed total acidity running 38, 25, 18 and 12, with free HCl 28, 14, 8 and 6. The x-ray report said: "There is a definite hour-glass constriction of the stomach with a large out-pocketing defect on the lesser curvature at the level of the narrowing." This patient was treated by rest, Sippy diet, alkaline powders and belladonna. In one month all her symptoms had disappeared, and she began to gain in weight. Two years later, when last seen, she weighed 115 lbs. and felt perfectly well if careful about her diet.

A second case is interesting because it shows how a patient may survive practically all the complications of chronic gastric ulcer and die at last from another cause.

A woman, aged 72, had been troubled by her stomach as far back as she could remember. Years previous she twice had serious hæmatemesis, but none since an emergency operation for perforation, fifteen years before this present consultation. For several months recently she had suffered severely from pain in her stomach by night as well as by day, coming on one to four hours after food, no longer relieved by soda as it used to be. There was no vomiting.

This patient was found to have a palpable mass in her upper abdomen, resistant and tender. Gastric analysis showed a deficiency of free HCl, total acidity running 10, 20, 16 and 8, and free HCl 0, 10, 8 and 0. The x-ray report stated: "Stomach is hour-glass in shape with a large out-pocketing defect about the middle of the lesser curvature; definite fixation of the lesser curvature both above and below the large ulcer crater." This patient on routine ulcer treatment by rest, Sippy diet, alkaline powders and belladonna, gradually improved so that two months later she reported she was free from pain and felt well. She continued to be free from stomach disturbance until her death six months later from a cerebral hemorrhage.

## CHAPTER VI

### CANCER OF THE STOMACH

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Cancer attacks the stomach more frequently than any other organ in the body. But on the other hand among the many causes of disturbance of gastric function this type of disease is fortunately not the most common. It is always a possibility whenever chronic indigestion is the complaint. It should always be the first thought and its presence suspected in every case when the patient's story is of "stomach trouble". But of the many who seek advice for such disorders, few will prove to have as the cause this most serious form of pathological change. It is more common in men than in women, and while most cases are seen in patients past 40, it does occur at times in subjects below that age. The old teaching that only in middle age or beyond is cancer of the stomach encountered is, therefore, incorrect and no longer should be trusted.

Cancer of the stomach does not always originate at the same site, and where it is located determines largely the character of the symptoms produced and all the signs upon which diagnosis depends. It may develop at the cardiac end, in the middle portion of the organ or at the pyloric end. Furthermore it undoubtedly develops at times on the base of an old ulcer.

Thus there are four clinical groups to be considered in diagnosis. The history in each one of these is different from the other, but never from history alone can a trustworthy conclusion be reached. What is found by physical examination, by gastric analysis and by x-ray films likewise differs so much in these four groups that only by considering each separately can there be a proper understanding. There is no description of cancer of the stomach that covers all cases.

#### CANCER AT CARDIAC END OF THE STOMACH

Cancer originating at the cardiac end of the stomach may produce no symptoms at all of sufficient importance to lead the patient to seek advice until it is large enough to interfere with swallowing of food. The common story is that the patient began to notice first difficulty about getting coarse foods to pass. At intervals a piece of meat or a wad of bread, not well masticated, would stick and cause discomfort. This occurred with increasing frequency as time went by. Gradually this difficulty is noted with softer foods as well. Then vomiting becomes a symptom, or rather regurgitation, for the food comes back into the mouth even before it has had time to reach the stomach. The significant feature about this symptom is the promptness with which it occurs after eating, almost immediately, while still at table and not an hour or two later. As a result of this persistent food rejection, the patient loses weight, strength and color, so that emaciation, weakness and pallor gradually become a part of the clinical picture. Pain, if it is noted at all, is described as a dull, heavy ache rather than the cramps and sharper colic caused by pyloric obstruction. This pain is produced by pressure on surrounding parts by the tumor and is referred usually not to the epigastrium, but higher up

beneath the sternum, or to the left costal margin, the left axilla or the left back between the spine and the scapula. It is possible for such a tumor to develop at the cardiac end of the stomach without obstructing the orifice where the œsophagus enters. In such cases dysphagia will not result to give early warning. Then the history is one simply of failing health with no symptom to identify the cause except the dull pain in the back, left axilla or under the sternum with loss of appetite, weight, strength and color.

Physical examination, while it reveals weight loss, anæmia, low blood pressure and other evidences of poor nutrition, usually proves absolutely negative in this group as regards the abdomen. A large tumor at the cardiac end of the stomach may completely elude discovery by the ordinary methods of inspection and palpation, because it lies so high up under the diaphragm.

Gastric analysis likewise is practically useless in these cases. Either a test meal can not be swallowed, or else the tube can not be passed beyond the obstruction in order to obtain material for the test. The very fact, however, that a small tube, such as the Reh fuss, can not be made to enter the stomach is highly significant of an obstacle at or below the cardiac orifice. If it does pass and contents are obtained, they usually show no free HCl and abundant mucus, for the presence of such a growth quickly disturbs secretion and leads to development of a chronic gastritis.

Even x-ray examination gives little information except that there is obstruction at the cardiac orifice with dilatation of the œsophagus above it. What barium does find its way through usually is not enough to outline the stomach or to demonstrate the large defect at the fundus. The œsophagoscope has long been used to determine whether the obstruction,

recognized clinically, is situated in that tube or below its junction with the stomach. The gastroscope will have its value limited in such cases by the difficulty in passing it beyond the obstructing growth. The use of either of these instruments is not without danger, because it may perforate œsophageal or gastric wall made unusually friable by necrotic neoplasm. Neither one should ever be employed for diagnosis except by one thoroughly experienced in their use.

In differential diagnosis the only question of importance is whether the obstructing growth is in the œsophagus above the cardiac orifice or in the stomach below it. After all it does not matter much, for in either situation the growth is so inaccessible that its successful removal is practically impossible. Treatment has been recommended in these cases by perforated metal bougies, passed on a guiding thread swallowed hours previously, and sometimes this succeeds in keeping the stricture patulous and in prolonging life for weeks or months. But it does not cure, and sooner or later death is inevitable from starvation.

A man, aged 74, complained that for six months previous he had lost his appetite completely. He had not eaten meat because he could not get it down. Other food, semi-solid, he could swallow, if he took a hot drink to help it along. Frequently what he ate would come back again, but if it did go through, it caused no distress. Recently he had more trouble than ever about swallowing. He had no pain at all but now could take no food but liquids and consequently had failed rapidly in weight and strength. No abnormality whatever was found in the abdomen on physical examination except epigastric tenderness. A tube could not be passed into the stomach for gastric analysis, but from the pouch above the stricture a quantity of "swill-barrel" material was washed out, consisting of retained and decomposed food. X-ray examination showed organic

stricture at the cardia with dilated œsophagus above it. Attempts were made to pass dilating bougies on a swallowed thread as a guide but without success. They caused the patient so much discomfort, he would not tolerate them. Finally, to save him from starvation, if possible, a gastrostomy was done. This disclosed an immense tumor of the cardiac end of the stomach, running down on the lesser curvature, with many metastases in intestine and omentum. The patient died two weeks later.

### CANCER IN MID-PORTION OF THE STOMACH

Cancer originating in the mid-portion of the stomach, on the lesser curvature, anterior or posterior wall, may become extensive and inoperable before it causes sufficient disturbance of digestion to persuade the patient to seek medical advice. The symptoms may all seem vague and indefinite and apparently of no importance, such as decreased appetite, heaviness and distress after food and belching of gas. What is more likely to attract attention is a deterioration of general health without known cause. The patient complains that gradually strength has failed, energy and endurance have decreased, pallor has replaced previous good color, and weight has been lost. All this may go on for several months before advice is sought. There is no increasing dysphagia such as characterizes neoplasm at the cardiac end of the stomach, no persistent vomiting such as sooner or later results from neoplasm at the pyloric end and usually no pain of consequence.

*Physical examination* only too often reveals, by the time the patient concludes to consult a physician, a palpable tumor. This is found in the epigastrium or descending from beneath the ribs into the right or left hypochondrium. It may be felt as a nodule or a hard ridge or an irregular mass. Occupying the midportion of the stomach and thus lying high up in the

abdomen, the tumor may escape observation unless special effort is made to detect it. Palpation, while the patient sits erect and leans forward or palpation while the patient is lying down but turns to one or the other side may bring such a tumor within reach. No tumor may be found even when one exists, because it is fixed beneath the ribs, or because it is diffuse and not well localized. In such cases it sometimes comes as a distinct shock to the diagnostician to see the extensive defect disclosed later on by x-ray films. Unfortunately the failure to find a tumor by physical examination does not prove that the patient has none, while the discovery of one means a late diagnosis and cancer far advanced.

*Gastric Analysis.* — When cancer has invaded any part of the stomach wall, the fasting contents are always more or less abnormal. Nevertheless when the neoplasm occupies the body of the stomach and does not involve its inlet or its outlet, nothing characteristic may be found. The material obtained may show nothing unusual except diminished secretion and excess of mucus such as habitually is found in chronic gastritis. Fractional analysis in response to a test meal also gives variable results, but almost always secretion and motility are gradually impaired. Complete absence of free HCl, however, formerly taught to be a diagnostic sign, should not be expected, because it may not occur at all or only late in the course of the growth, and because achlorhydria may be associated with so many other diseases besides cancer, that its discovery proves nothing without additional evidence. Hyposecretion is the rule in the group of patients now under consideration and impaired motility, as shown by delay in the stomach's emptying time and poor nutrition. Interference with these two important functions is always significant but in no way diagnostic. Gastric

analysis, therefore, while a valuable means of acquiring information, can not be relied upon to furnish conclusive evidence by itself as to whether cancer of the stomach is or is not present.

*X-ray Examination.* — In every case of chronic indigestion, no matter what the other methods of investigation show or do not show, fluoroscopic examination and x-ray films of the stomach are indispensable. Particularly, when suspicion has been aroused that cancer may cause the symptoms, no reliable conclusion can be reached without this aid. When a tumor is palpable in the upper abdomen, x-ray films tell whether it originates in the stomach, and if so, what part of the wall it involves. When no tumor is discovered by physical examination, it may nevertheless appear as a filling defect in a part of the stomach that could not be explored by palpation. If no such defect is shown by x-ray examination, usually it is justifiable to conclude that cancer is not the disease causing the symptoms, no matter what the history or the gastric analysis suggest. If the symptoms persist, however, in spite of treatment, after one month x-ray examination should be repeated; for a small growth on the stomach wall, soft and non-protruding, such as adenocarcinoma, can be present early in its course and produce no defect in the x-ray films, and the same is true of a flat scirrhus type. The x-ray examination is not infallible, and error may occur in spite of every skill and care on the part of the technician. On the other hand, a defect reported by the x-ray consultant of such a character that it is interpreted to mean cancer of the stomach, may not appear at all when laparotomy is done and the organ explored. These possibilities exist and must be recognized, but in general what this method of investigation adds to the other evidence is in the great majority of cases entirely trustworthy.

It is just in the few cases where x-ray examination is indefinite, or where the report it supplies does not quite fit the other evidence obtained by history, physical examination and gastric analysis, that great assistance should hereafter be furnished by *gastroscopy*. The exact appearance of a growth in the stomach wall, the ability to obtain a bit of tissue from it for biopsy, the added proof thus afforded as to whether a chronic ulcer has become malignant, and the ability to decide by direct inspection whether the pathological change in a stomach is cancer or chronic gastritis, when history suggests the former, but x-ray films fail to show a defect, all these advantages will be supplied hereafter as experience with the gastroscope becomes greater and its use more widespread.

*Diagnosis.* — The only way to avoid over-looking cancer of the stomach is to keep the possibility always in mind. Not only when the complaint is of chronic indigestion but also in all the so-called “run down conditions” characterized by loss of strength, color, weight and energy, must cancer of the stomach be considered. This is particularly true in the group where cancer originates in the mid-portion of the stomach rather than at either end. Even in these patients the picture ultimately becomes so striking that the diagnosis is obvious, but then it is too late for the surgeon to be of service. Early recognition affords the only hope. When the patient’s story is of failure of general health more than of digestive symptoms, when no tumor is found, when gastric analysis shows nothing more definite than impairment of secretion and motility, then decision rests upon what x-ray examination discloses. If a definite filling defect is shown in the mid-portion of the stomach, no further doubt can remain, and not infrequently this is found when the patient’s physical condition did not seem to

warrant it. If x-ray examination does not show any such defect, and no other cause can be found for the patient's complaints, careful observation should follow, and x-ray examination should be repeated a little later on.

The disease most often confused with this form of gastric cancer is *pernicious anæmia*. Its prominent symptoms are weakness and weariness, pallor, lack of appetite and moderate weight loss with occasional upsets of the stomach and vomiting as one feature. Physical examination of the abdomen furnishes no evidence to explain this history. Gastric analysis shows achlorhydria as one of the constant and essential signs. X-ray films reveal no gastric defect. The diagnosis rests on examination of the blood, which presents a peculiar and characteristic picture. But on the other hand cancer of the stomach originating in its mid-portion also produces anæmia of greater or less degree, and this may resemble the blood picture of pernicious anæmia so closely as to be mistaken for it. Particularly when physical examination is negative and x-ray films fail to show any abnormality the case may easily be considered one of primary rather than of secondary anæmia. Further time for observation, the patient's reaction to specific therapy by liver extract and repetition of the x-ray examination later on will ultimately make the diagnosis clear. The association of cancer of the stomach with pernicious anæmia apparently is rare. Dr. Madelaine Brown, who reviewed the records of all cases of pernicious anæmia that came to autopsy in the Boston City Hospital, the Peter Bent Brigham Hospital and the Massachusetts General Hospital, one hundred and fifty in all, reported (New England Journal of Medicine, Vol. 210, p. 473, 1934) that only one showed cancer of the stomach.

A man, aged 76, sought advice about progressive weakness. He had been ailing for six months with loss of strength and energy, dizziness and giddy spells. He had grown noticeably pale, had been told he was anæmic, had been treated by liver extract but had failed to improve. He had a good appetite, no distress after food, no nausea or vomiting. There had been no loss of weight. No mass was palpable in his abdomen or any other abnormality found there. X-ray films of his gastrointestinal tract, made six weeks previous at the direction of another physician, had demonstrated no defect in the stomach. His blood count, however, showed only 54 per cent. hemoglobin, 2,950,000 red cells, with very definite abnormality in their size and shape and color, and an average diameter of 7.9 micra. By gastric analysis complete achlorhydria was found. There was no occult blood in the stool. In spite of the very suggestive blood count, cancer of the stomach was suspected, and x-ray films were repeated, two months after the first ones were made. This time they showed a large ulcerated area on the lesser curvature, 3 cm. in diameter. Treatment did not produce any improvement, and six weeks later a mass was palpable in the epigastrium. This gradually increased in size and became very tender. The blood did not improve to any extent in spite of liver extract. Repeated counts from time to time never differed materially from the first one. As time went by, the patient's weakness became more and more pronounced. A third x-ray examination of the stomach made six months after the second showed "an extensive ulcerating lesion on the lesser curvature, along its distal third, presumably carcinoma". He died one year from the time he first came under observation. Toward the end he vomited frequently a coffee-ground material and suffered much pain.

There is no excuse for failing to recognize cancer of the stomach, once the patient ceases to procrastinate about seeking advice. If in every instance a careful and complete history is taken, involving all the various systems of the body and not simply the digestive, if the physical examination is thorough

and painstaking, including the rectum and genital organs as well as the viscera of thorax and abdomen, if laboratory tests are made of blood, urine, stomach contents and stool, and if fluoroscopy and x-ray films are employed as a routine part of the collection of data, then there is little likelihood that cancer of the stomach will ever fail to be recognized, no matter what its site or duration may be. Here, as elsewhere, the rule holds good that error in diagnosis is the result of incomplete observation and investigation.

*Treatment.* — As soon as the diagnosis is made, the patient should be referred to a surgeon, for operation offers the only chance for cure. There are only three situations that justify medical treatment instead; first, when the diagnosis is uncertain at the outset, and therapy becomes an aid in settling it; second, when the patient refuses operation that has been advised; and third, when the surgeon decides or exploratory operation proves that successful removal of the growth is impossible. The first situation, for instance, may arise when chronic gastritis, chronic gastric ulcer or pernicious anæmia appear possible explanations instead of malignant disease. If appropriate treatment for the condition supposed to be present does not cause improvement in a month, further investigation should be made with repetition of all previous tests. The second situation may conceivably result when extensive growth involves the midportion of the stomach, and its removal would mean practically a subtotal gastrectomy. After this is explained and the risks of operation as well as the possibility of early recurrence are made clear, the patient may well conclude to let matters take their course. This decision has been reached several times by physicians themselves afflicted by this disease. The third situation is only too often the consequence of

a late diagnosis. Cancer of the stomach with metastases to the liver or intestines discovered by exploratory laparotomy is hopeless for cure.

In all these cases, as well as in the one where even exploratory laparotomy is not accepted, the ingenuity of the physician is taxed to prolong life and relieve suffering. The only plan to follow is a symptomatic one; to keep the patient nourished by soft foods, liquid or semi-solid, in small quantity at short intervals, to aid digestion by hydrochloric acid, pepsin, pancreatic extract or by predigested foods, to check anæmia by liver extract and iron, to prevent pain by opiates, administered in sufficient quantity and often enough to accomplish this without the usual fear of establishing a bad habit by such drugs. Above all in these cases, where he is fighting a losing battle with no chance to win, the physician's mental attitude becomes of the utmost importance. He must remember that he has it in his power to keep his patient cheerful and contented, even though he can not save life, and to make the invalid happier by his visits, even though he can not cure his physical ills. Should the patient ever be told his condition is hopeless? This is a question in ethics which must be answered according to the circumstances of each case and the temperament of each individual. To the writer it seems that in some instances deception is justifiable, provided the family or some trusted friend is made aware of the facts and approves evasion of the whole truth.

#### CANCER OF PYLORIC END OF THE STOMACH

Cancer of the stomach originating at the pyloric end is the most common and familiar type of which the symptoms usually are recognized early. In a patient, as a rule over 40

but not necessarily so, who previously has been well and had no digestive ailment, appetite begins to fail, and persistently the desire for food decreases. Distress comes on soon after eating, a sense of fulness and weight and oppression, ultimately becoming real pain, dull or severe, felt in the epigastrium. There is more or less constant belching of gas. With distress frequently comes nausea, and with increasing frequency this is followed by vomiting. Sometimes when vomiting is not spontaneous, it is induced, because emptying the stomach gives relief. From loss of desire for food, fear to take it because of the discomfort it causes and frequent loss of it by vomiting, the patient slowly becomes thinner, weaker and paler. In many cases the vomitus contains brown flakes or shreds, due to blood that has oozed from an ulcerated surface and has been partly digested before it is evacuated. This is a late sign, however, and should never be awaited for diagnosis. Long before it is seen, suspicion should be aroused and thorough investigation made. Only too often, however, the patient is at fault for the delay, waiting until a lump is discovered in the abdomen before seeking advice and relating the story that has preceded. Pyloric obstruction occurs early in this group and leads to further tell-tale manifestations, such as amounts vomited apparently in excess of amounts swallowed, substances recognized in the vomitus one day that were eaten a day or more before, and a foul odor of the vomitus, due to decomposition of food and to discharges from a necrotic ulcerating area. All these details indicate pyloric obstruction but do not identify the cause. For this, other methods of investigation are required.

*Physical Examination.* — Originating at the pyloric end of the stomach, below the level of the rib cage, cancer frequently presents a visible and palpable tumor when first the abdomen

is examined. If the patient, lying on the bed or examining table, is placed in a good light, the tumor often can be seen descending, when a full breath is taken. Or if it is not visible, it can be palpated in the epigastrium or descending from beneath the ribs in the right or left hypochondrium. It varies in size and in shape as well as in situation. Usually it is tender to touch, and this has often led the patient to note its presence even before advice is sought. As a rule it is not fixed and moves or can be moved about freely in the abdomen, changing its position on deep inspiration or on change of posture by the patient. It may be noted at one examination and not at the next. If not discovered when the patient is lying on the back, it may be distinctly observed when the patient is sitting or standing, leaning forward. Evidence of pyloric obstruction may also be noted, as in pyloric ulcer, such as a visible bulging area outlining the stomach with a peristaltic wave running across it from left to right. In some cases of dilated stomach, due to food retention from pyloric obstruction, it may be necessary to wash out the contents before a tumor is palpable. All of these signs, however, are distinctly late evidence of pyloric cancer, and diagnosis could be made long before they appear, if only patients would present themselves sooner for examination.

*Gastric Analysis.* — Stomach contents before obstruction has occurred usually show nothing characteristic. But obstruction may take place early in this group and may be noted by gastric analysis even before clinical evidences of it appear. Thus the fasting contents are found to be larger in amount than normal, above the average 50 c.c., frequently above 100 c.c.; the material is thick and viscid; it may show fragments of retained food visible to the naked eye; analysis gives a low total acidity

with no free HCl; and usually mucus is present much in excess of normal amounts. Even quite early in the history, before vomiting has occurred at all, the fasting stomach contents may be blood-stained or show brownish flakes. Fractional analysis after a test meal gives variable results, depending upon whether obstruction of any consequence has taken place. But hypersecretion, normal secretory curve, or hyposecretion are all compatible with a diagnosis of pyloric cancer in its early stages. Secretion gradually fails, however, as the neoplasm grows, due largely to the gastritis induced by retention and decomposition of food, as obstruction becomes a feature of the case. Complete anacidity is a late result, and to withhold diagnosis until this is found is a fatal error, long emphasized by many authorities.

*X-ray Examination.* — The picture presented by pyloric cancer varies because gradually increasing obstruction modifies the changes produced by the growth itself. Sometimes the films show a ragged, irregular defect at the pyloric end of the stomach, as if the tissues had been gnawed away by a rodent. Sometimes there is visible a distinct ulcer crater due to necrotic changes in a carcinomatous nodule. Sometimes all that is visible is a blunt pyloric end with lesser and greater curvature bulging around it, due to obstruction. Finally, at times there is seen a smooth pyloric narrowing or a long tubular pyloric end of the stomach with no peristaltic waves passing over it, the so-called “goose-neck” stomach. All of these abnormalities are highly significant. They do not by themselves make the diagnosis, but they can never be disregarded, and taken in connection with other evidence they assist greatly in the production of a proper understanding.

*Diagnosis.* — Unfortunately, in most cases of cancer originating at the pyloric end of the stomach, recognition is easy.

This is because the patient appears so late. A palpable abdominal tumor and achlorhydria are late events in the course of the disease, and recognition of malignancy should long precede these manifestations. The chief reliance for early diagnosis should be placed on x-ray examination. If this were done sooner, while there are no symptoms except those of persistent indigestion, with no pain, no vomiting, no palpable tumor, no decided reduction in gastric secretion, no noticeable loss of weight, strength and color, earlier diagnosis could undoubtedly be made, and better results from surgery could be expected. However, the fault for delay does not lie so much with the physician as with the patient, who postpones seeking an explanation of his "stomach trouble" until it is too late.

The two other organic diseases of the stomach most often confused with cancer are chronic gastritis and chronic ulcer. Attention has already been called in the discussion of *chronic gastritis* to the fact that it may resemble early cancer of the stomach in its symptoms, particularly in the lack of actual pain and the presence only of discomfort and distress after meals, in the absence of any distinguishing signs on physical examination, in the findings by gastric analysis, and in the normal contour of the stomach shown by x-ray films. In this group of cases it is always well to be suspicious, especially if the patient's illness is comparatively recent, to watch closely for one month at least the developments that take place while treatment is directed to chronic gastritis, and to repeat the tests of gastric secretion and the x-ray films before the patient is dismissed from observation.

*Chronic gastric ulcer* usually has such a long-standing history, so characteristic and so different from that of cancer, that this alone suggests the diagnosis. The pain caused by ulcer, as

a rule, is more intense than that caused by cancer, more localized, relieved entirely by taking food and for a variable period afterwards, but returning after the lapse of one, two or more hours with all its former intensity, while the pain in cancer has no such periods of intermittence or rhythm and does not present such associated symptoms as sour stomach with burning, water-brash, highly acid and irritating vomitus. The hæmatemesis of cancer usually is small in amount and peculiar in appearance, because slow oozing of blood from an ulcerated surface results in its partial digestion before it is vomited. Thus the material evacuated consists of brown flakes and looks like coffee-grounds. Ulcer on the contrary usually produces a discharge of bright red blood of greater amount because a small or large vessel in its floor has been opened. But this difference is not always observed and a large hæmorrhage sometimes may be caused by cancer, as the following case history illustrates.

A man, aged 66, got up one morning and went to the toilet. A little later he was found on the floor unconscious, having vomited blood and passed it by bowel in large quantities. For some time before this incident he had felt well, without pain or indigestion and with no discomfort except occasionally a sensation as if his food clogged in his stomach. Following this he had no further bleeding, no indigestion, no pain, but felt very weak from loss of blood. Yet a palpable sausage-shaped mass was found in his right hypochondrium, gastric analysis showed complete anacidity, and x-ray films disclosed an irregularity of the pyloric end of his stomach which made it tubular in shape. He died six months later, and autopsy confirmed the diagnosis, showing a large scirrhus carcinoma at the pyloric end of the stomach.

Physical examination may be negative in either ulcer or cancer except for localized tenderness over the epigastrium or

one hypochondrium. Even when a palpable, tender mass is found, it may be due to a large indurated, inflamed ulcer of long duration rather than to carcinoma, and other evidence obtained by gastric analysis and x-ray films must be taken into consideration before a conclusion is reached. Stomach contents show abundant acid in typical ulcer, and hyperchlorhydria is characteristic, while secretion is deficient in advanced cancer and ultimately there is none at all. This rule does not always work, however, for in chronic ulcer deficient acid and in cancer an abundance may be found in exceptional instances. Therefore, it can not be too often repeated that gastric analysis by itself is never sufficient for diagnosis. Finally, the x-ray examination is depended upon to decide whether ulcer or cancer is the pathology concerned. There are cases, however, in which even this method of investigation is unable to determine whether a defect seen at the pylorus or on the lesser curvature near it is produced by ulcer or cancer. There remains also a fourth group of cases of cancer of the stomach, yet to be considered, in which a lesion originally ulcer has become cancer, and this possibility, even though it be remote, as some authorities claim, must nevertheless be kept in mind. Some of the difficulties encountered in the differential diagnosis between the two are illustrated by the following case.

A woman, aged 46, complained that for five months previous she had no appetite at all, but pain in the pit of her stomach, sourness and burning, water-brash and frequent vomiting which relieved her distress. She was pale and emaciated and a distinctly palpable mass, like a hard ridge, was found in her upper abdomen. Gastric analysis showed a hyperchlorhydria running as high as 95 total acidity with 45 free HCl after the Ewald meal. The stool showed occult blood. X-ray examination disclosed a defect involving the pyloric end of the stomach with some fixation along the lesser curvature and def-

inite obstruction. By combining all the evidence obtained a diagnosis was made of chronic ulcer. On routine treatment for this, she lost all her symptoms, gained ten pounds in weight, and the palpable mass disappeared. Three months later, however, because pyloric obstruction had recurred and increased, exploratory operation was done. A large indurated ulcer on the lesser curvature near the pylorus was found and resected. Pathological examination of the tumor showed it was cancer. She died six months later from recurrence with peritoneal metastases and ascites.

It is remarkable how far advanced cancer of the pyloric end of the stomach can become before it produces symptoms of sufficient consequence to attract the patient's attention. Then it is often too late to permit surgery to be of service.

A man, aged 64, was positive that he had no stomach trouble until about a month before he sought advice. For that length of time he admitted his appetite had been poor, occasionally he had been nauseated, and several times during the month he had vomited, mostly in the morning on rising, just a little fluid but no food. There was much belching of gas after meals, occasionally with sour eructation. He felt a little heavy and uncomfortable after eating but insisted that he suffered no real pain. He had lost no weight or color and felt as strong as ever. No tumor was palpable in the abdomen. The Ewald test meal showed complete achlorhydria. X-ray films demonstrated that the pyloric half of the stomach was tubular in shape, and no peristaltic waves passed over it; the stomach emptied rapidly; there was no irregularity or raggedness in its outline. This patient felt so well that he scorned exploratory operation and went about his business as a ship captain. Two months later, however, progressive failure of health necessitated operation in another port, and this revealed a large scirrhus carcinoma of the stomach, involving approximately the pyloric half of its substance. He died shortly afterwards.

Remarkable as is the fact that extensive scirrhus carcinoma may involve a large part of the pyloric end of the stomach

without producing any threatening symptoms, even more remarkable is the length of time such pathology may persist before it causes disturbance of health sufficient to demand action. The following case history illustrates this fact.

In February 1917 a man, aged 51, sought advice for pain in the pit of the stomach after eating, observed ever since the preceding August. Appetite was good, but pain in the stomach, though present almost continuously, was always worse soon after eating. He felt nauseated at times and frequently had induced vomiting because emptying the stomach gave relief. He had lost weight, about 20 lbs., in the preceding six months since his ailment began. Always previously he had been well, with no digestive trouble. On physical examination no palpable mass was found in the abdomen. The fasting stomach contents amounted to 250 c.c. Gastric analysis showed complete achlorhydria. The Wassermann reaction at this time and subsequently was always negative. On x-ray films of the stomach the report stated "no evidence of neoplasm is found". On treatment by soft diet with dilute HCl after meals he lost all his distress, gained steadily in weight and by April felt so well that he was dismissed from observation.

He remained well for one year. Then in April 1918 he reported that there had been slight recurrence of pain after taking two glasses of cold beer. The abdomen again was negative on physical examination. With care once more about eating and drinking, his distress all left him, and after a few days he disappeared again from observation.

This patient's third report for advice was in September 1919, a year and a half later. Then he complained that for three or four months past he had pain again in his stomach after eating, becoming gradually more acute and persistent. He was constantly hungry and no food was lost by vomiting, but he could not eat much at a time. At this visit the abdomen was found more tender and rigid in the right hypochondrium than in the left, but there was no palpable mass. Gastric analysis again showed complete absence of free HCl.

This time the x-ray technician reported; "the outline of the stomach is regular as far as the region of the pyloric antrum, but in this portion there is a definite persistent narrowing, and at six hours considerable delay in emptying." The patient, however, would not accept the suggestion of probable carcinoma or the advice that exploratory laparotomy ought to be done. He was again placed upon a soft diet and a mixture to aid digestion, and after one month felt so well that after all the diagnosis seemed doubtful.

His fourth return for report about his condition was in November 1920. During the year since he was last seen, he said he had felt well except when careless about his diet. He desired and would accept no further investigation at this time.

Another year went by before his fifth visit, in October 1921. Meantime his wife had insisted upon his consulting another physician for diagnosis, who likewise reported finding complete achlorhydria and a narrowing of the pyloric end of the stomach. Cancer of the stomach was the verdict, but the patient refused to accept it, saying he had been told the same thing two years before, had remained well since if careful about his diet and therefore could not have cancer.

His sixth and final return for advice was in February 1922, five years after his first. By this time he complained again of pain after eating, complete loss of appetite, nausea and vomiting each morning on rising and more disturbance of general health than ever before. His pain became more and more severe, there was marked tenderness over the stomach, he lost weight and strength and became anemic. A forcible peristaltic wave was now visible across the stomach. X-ray examination, repeated in August 1922, reported: "no irregularity in the outline of the stomach up to the pyloric third, which showed a large organic defect; delay in emptying time so great that even at forty-eight hours half of the meal was still retained." Now consenting at last to surgery, this patient was operated upon a few days later. The surgeon found a large carcinoma of the pyloric end of the stomach, necessitating removal of approximately one-half the organ. The patient died a few days later of

postoperative pneumonia. Pathological examination of the tissue removed proved it to be adenocarcinoma.

*Treatment.* — For cancer of the stomach originating at the pyloric end, as for cancer at any other site, the only hope for cure lies in its removal by surgery. But if operation is refused by the patient or found unjustifiable by the surgeon, it then becomes necessary for the physician to do what he can to keep the patient comfortable and to prolong life. In addition to all the measures suggested for management, when cancer involves the mid-portion of the stomach, it becomes necessary in the group involving the pylorus to prevent the slow starvation that results from obstruction. For this purpose morning lavage with hot bicarbonate of soda solution to prevent food accumulation and to keep the stomach clean helps also in overcoming the chronic inflammation that adds to the edema and spasm at the pylorus. For the same reason, tincture of belladonna is of value, because it assists in relieving pylorospasm. It can be given alone or in combination with a sedative, such as a few drops (5 to 6) of the deodorized tincture of opium. If these drugs administered by mouth cause nausea, the aqueous extract of opium with the extract of belladonna can be used in a rectal suppository.

#### CANCER OF STOMACH ORIGINATING ON ULCER BASE

There is no difference of opinion now about the possibility that cancer of the stomach may develop on an ulcer base, but there is great difference of opinion about how often this occurs. In fact no question in the discussion of gastric disease has caused more controversy than this. Some authorities take the extreme view that all cancers of the stomach begin as ulcers and that commencing malignant degeneration can be

detected by the pathologist in practically every chronic ulcer excised; others admit that a small percentage, five to ten, start in this way; between these two views, there are all sorts of estimates of frequency.

It is the clinical history in any case under investigation that first creates suspicion. The patient gives an account of a chronic digestive disturbance going back for years, with the typical story of chronicity, periodicity, rhythmical sequence of events and character of symptoms that so commonly means ulcer. But recently, in the latest attack, it is recognized that what is taking place is different from what has occurred before. There is more constant pain, not relieved by taking food but rather increased by it; loss of the usual keen appetite, progressive loss of weight, strength and color, to a degree that no previous attack has ever produced. Physical examination may reveal no more than has always been found before, decided tenderness over the upper abdomen, in epigastrium or one hypochondrium. Even if a palpable tender tumor is found, this is not uncommon in a chronic pyloric ulcer at the time of active inflammation with œdema and does not inevitably mean cancer. Evidences of pyloric obstruction supplied by history and the presence of a peristaltic wave across the stomach may be attributed with equal propriety to either ulcer or cancer. Hæmatemesis of any appreciable amount is more common in ulcer than in cancer, but exceptionally a large hæmorrhage may be the first prominent sign of cancer, and malignant change in an ulcer does not prevent it from bleeding as it might have done before. Occult blood in the stool may be due to either one. As regards gastric analysis, when ulcer has long persisted, an excess of free HCl may continue to be secreted even after malignant change has taken place. Even x-ray examination is

not always conclusive in differential diagnosis, for many times the appearance of the lesion is simply that of chronic ulcer. A large ulcer on the lesser curvature always creates suspicion that it is malignant or may become so. But not infrequently after routine ulcer treatment, even these become quiescent, smaller and no longer troublesome. Pyloric ulcer likewise may, by its appearance, lead the conscientious x-ray technician to suggest the possibility that it has become cancer, but he can not make the diagnosis from x-ray appearances alone. The only rational procedure in such a dilemma, after weighing all the other evidence as well as that supplied by x-ray examination, if doubt still exists, is to treat the patient for ulcer and then judge by clinical progress and by repetition of x-ray films whether to go on with this plan or to have the ulcer excised so that the pathologist can decide the matter. But ulcers, already malignant, may respond temporarily to treatment by diet and medication with improvement in all symptoms and with decrease in the size of the ulcer crater in x-ray films. This, however, is the exception and not the rule.

That in some instances at least cancer of the stomach may originate on the base of a chronic ulcer, the following case histories seem to prove. In any event, they show the facts upon which dependence is placed in reaching such a conclusion.

1. A man, aged 41, following a long history of indigestion for years, had his pylorus excised for chronic gastric ulcer. The excised tissue showed beginning malignant degeneration at the ulcer's edge. Two years later recurrence of indigestion with severe pain and loss of weight prompted a second operation, when it was found that he had a large, hard, nodular tumor of the stomach, extending to and involving the pancreas.

2. A second patient, aged 55, had been ailing at intervals for fifteen years with sour stomach, burning, water-brash and belching,

all making their onset two or three hours after eating. Then about a year before seeking medical advice he began to have severe pain not experienced in any former attack and to lose progressively in weight. Operation revealed a carcinoma involving the stomach to a degree that made its successful removal impossible.

3. In a third patient, aged 41, the story was that for years past he had attacks of indigestion and sour stomach, lasting about two months at a time, then gone again for months. The present attack, lasting for over one month, was characterized by more intense pain than experienced in any previous one, relieved for an hour or two after eating, then recurring and persisting until more food or else bicarbonate of soda was taken. Nausea accompanied this latest attack, and for two weeks he had vomited nearly every day. He had never vomited blood but for a week had noticed that his bowel movements were always black. The upper abdomen was very rigid and tender, but no tumor could be discovered. On inflation with CO<sub>2</sub>, a definite peristaltic wave ran across it from left to right. Gastric analysis showed a total acidity of 80 with free HCl 40 after the Ewald test meal. This was before the days of routine x-ray examination, but even without this aid the diagnosis of chronic gastric ulcer could be reasonably inferred. Treatment, however, by diet and medication did not relieve his pain at all. He vomited frequently, and the fluid vomited at times was black. Pyloric obstruction increased, as shown by the vomiting of retained food in large quantities with more found by lavage. Nevertheless gastric secretion remained high with total acidity 72 and free HCl 52. Operation was finally made necessary by obstruction supposed to be due to chronic gastric ulcer. What was found was a hard, irregular, nodular mass extending along the lesser curvature from the pylorus. No attempt was made to remove it because a metastatic nodule was discovered in the liver.

From the foregoing account of cases, as well as from a number of other similar ones observed, several inferences seem justified. First, there is no doubt that cancer of the stomach

does originate at times on an ulcer base, though probably not very many begin so. Second, even before any symptoms arise to make cancer suspected, excision of a chronic ulcer supposedly benign may show that malignant degeneration has already taken place. Third, by the time the patient comes for relief of an attack apparently similar to others he has had before and always cured previously by medical treatment, his ulcer may have become cancer so extensive it can not be successfully removed. History does not always tell when the change has taken place, nor does physical examination nor gastric analysis nor even x-ray investigation. Theoretically the gastroscope, by permitting a piece of tissue to be removed from the ulcer's edge for biopsy, affords the best means for settling the question before operation. But this can be effected only by the straight tube gastroscope and not by the flexible one, a procedure by no means a simple one or suitable for general use. The only certain way to avoid error would be to have every ulcer excised as soon as it is diagnosed, so that the pathologist could determine, but partial gastrectomy as a routine offers even greater risk to the patient than the possibility of malignant degeneration. Thus it seems best, all things considered, to adopt the plan already advised for the management of any doubtful case; treat the patient first for ulcer and then be guided by the clinical course and by the x-ray films as to whether surgery shall be employed or not.

CHAPTER VII

SYPHILIS OF THE STOMACH

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In the writer's personal experience no case of syphilis of the stomach has ever been observed, though diligent search for one has been made throughout a number of years. In approximately 260,000 admissions to the clinics at Stanford Medical School and Hospital no patient has ever been seen to whom this diagnosis could properly be assigned. It must be, therefore, that syphilis of the stomach is a rare disease. Most authorities agree about this fact. The few, who think it is not so unusual, do so because they demand less rigid proofs for its recognition. If histologic examination of tissues, removed at operation or at autopsy, is considered essential to establish diagnosis, very few cases have been accepted to date. But if history, blood Wassermann reaction, x-ray films and response to therapy are considered adequate criteria, the number of cases reported becomes much larger.

CLINICAL HISTORY

There is no typical account of disturbed gastric function that can be accepted as diagnostic of gastric syphilis. The

symptoms of which complaint is made are variable and do not correspond to any particular type of disease of the stomach. Pain is the one most often mentioned, appearing soon after eating, persisting until relieved by vomiting. This pain is described as aching, boring, burning or colicky. Its severity varies as well as its character. It is not relieved by food or alkalies, like that of ulcer, but vomiting, whether spontaneous or induced, does give relief until more food is taken. Hematemesis is rare but may occur. Loss of weight results in some instances because the patient fears to eat and because food taken is lost by vomiting. There may be exacerbations and remissions as in ulcer with periods of freedom from all symptoms for months at a time. The course thus is prolonged and may extend over years. The feature about the history that is most suggestive of syphilis of the stomach is its failure to correspond to that of any other type of chronic gastric disease.

### EXAMINATION

*Physical Examination.* — The peculiar pallor and anæmia, that characterize cancer of the stomach in its later stages, are not observed in this disease in spite of the usual lengthy history. There is no palpable tumor, and this fact is emphasized by those who have reported cases, because the history so often suggests cancer. Even when extensive deformity is shown later on by x-ray films, this usually is due to a widespread sclerosis rather than a localized nodular growth and is, therefore, not readily palpable.

*Gastric Analysis.* — The characteristic finding by this method of investigation is achlorhydria or at least decided hypochlorhydria, even after histamine. This has been reported most often and hyperchlohydria practically never. If the latter has

been found, later investigations have proved that the case was not anatomically syphilis of the stomach.

*X-ray examination* discloses a peculiar picture of deformity of the stomach, involving usually the pyloric end, but sometimes the entire organ to a greater or less degree. There is shrinkage and contraction, stiffening and decreased pliability, absence of peristalsis in the area involved, so that the stomach ultimately becomes a firm, tube-like structure with thickened, inelastic walls.

### DIAGNOSIS

Any long-continued disturbance of gastric function may mean syphilis of the stomach, whether the clinical history suggests chronic gastritis or ulcer or cancer, or resembles no type at all. But the chances are all against it being syphilis. If the blood gives a positive Wassermann reaction, this never justifies the assumption that coincident gastric disease is luetic, for syphilis in the blood does not prevent the development of some other pathological condition in the stomach. Physical examination of the abdomen usually is negative. Achlorhydria by itself fails to identify any disease. X-ray examination demonstrates deformity of the stomach suggesting scirrhus carcinoma, and in any doubtful case the chances are always in favor of cancer because of its much greater frequency.

At times, however, certain considerations may justify delay in recommending surgery, until the effects of anti-luetic therapy have been tried. These are the presence of a positive Wassermann reaction, the age of the patient below that usual for cancer of the stomach, a duration of symptoms over a period longer than malignant disease usually continues before a fatal outcome, a history corresponding perhaps in a certain degree to

that of ulcer but with persistent anacidity or sub-acidity found by gastric analysis, and no tumor palpable though extensive deformity of the stomach is shown by x-ray films. If to all these peculiar features is added improvement or apparent cure following specific treatment, it is natural to conclude that the real condition was syphilis of the stomach and to so record it. The fallacy about this is that syphilitics not infrequently develop some other disease entirely different from their primary one. Treatment helps the latter but does not cure the former.

### TREATMENT

When the evidence obtained by all available methods of investigation points to chronic gastritis or ulcer, but with a positive Wassermann reaction in addition, all the measures recommended for the treatment of these gastric conditions should be employed, but routine specific therapy should likewise be given. When there is reason to believe that a large gastric defect shown by x-ray may possibly be due to syphilis rather than to cancer, not only because the Wassermann reaction is positive but also because certain other suggestive details already described are found, it is justifiable to push routine specific therapy for a limited period before advising operation. But this plan must never be continued long unless improvement is definite. Even when this does seem to follow, the therapeutic test is not always reliable, for several reasons. First, in spite of vigorous anti-syphilitic treatment, which produces a negative blood Wassermann, the deformity of the stomach wall may persist and ultimately demand surgery for pyloric obstruction or hour-glass contraction. Second, a large benign ulcer with extensive scar tissue about it may improve greatly under specific treatment for syphilis, even though the latter

disease was never a factor in its etiology. Third, even a carcinoma may at the outset be benefited by a course of anti-syphilitic therapy, but only for a short time. In general, therefore, when doubt exists, it is wiser not to persist too long in medical treatment, but to advise exploratory operation, and if a neoplasm is found to have it excised and let the pathologist decide as to its nature.

## CHAPTER VIII

### POLYPS OF THE STOMACH

This is an extremely interesting subject in gastric disease and constantly becoming more so, as case reports increase and knowledge improves about methods of recognizing the condition. The fact that polyps may form inside the stomach is not a new discovery. Gastric polyposis was first described by Cruvelhier in 1833, over a century ago. Menetrier in 1888 made the first comprehensive study and detailed description of polyps of the stomach. After that case reports continued gradually to accumulate in medical literature, but all were observed only at autopsy until 1909. Then one was finally recognized during life, but only then by accident during an operation performed supposedly for cancer of the stomach. It is only in the past decade that increased perfection in x-ray technique and the construction of a practical gastroscope have made recognition of the condition comparatively easy. This newly acquired facility of discovering polyps in the stomach has demonstrated also how much more frequently they occur than was formerly supposed, though still they are by no means common.

Three types of distribution of such polyps are encountered, one multiple with many scattered over a large area or the entire wall, a second with a moderate number closely aggregated over a smaller area with a plaque-like base, and a third with only one large single polyp. In the multiple variety the number of these outgrowths may be two or three hundred or even more,

covering practically the entire interior surface of the stomach wall. In the second type, with a smaller number all located in one area of the stomach, the thickened base from which they arise may occupy any part of the stomach but more often is nearer the pyloric end than the fundus. The large single polyp may originate from any part but almost always is located at or near the pylorus. These tumors vary in size from a pea to a hazel nut. In color they are red or grayish red, in shape cylindrical or club-like, in consistency soft. They are highly vascular and bleed easily. The single polyp may be 2 or 3 cm. long, pedunculated or attached by a broad base. Multiple polyps have been observed more frequently in males than in females. They have been reported at all ages from 40 to 90 but with the largest number in the fifth and sixth decades. Their cause is obscure. They may be congenital or may develop as a consequence of chronic gastritis.

The *symptoms* that gastric polyps may produce are very variable. To begin with, there may be none at all, so that the presence of such benign tumors is never suspected until they are found at autopsy. Frequently they are silent over long periods of time, until ultimately some complication calls attention to them. There is never any definite syndrome that identifies them. The most constant manifestation appears to be epigastric pain, but this varies in degree from an indefinite discomfort to severe aching or cramps. Appetite usually is good, but anorexia may occur. Nausea and vomiting appear only when the polyps have formed a large enough mass to interfere with peristalsis, or when a single polyp has prolapsed so that it obstructs the pylorus. This last occurrence may annoy only when the patient is erect but not when lying down. Next in frequency to pain comes bleeding, shown by hæmatemesis or mæ-

lena or both. This is a common symptom because of the vascularity of polyps, and it may be the only one. The amount of blood discharged may be considerable, resembling the hæmorrhage of gastric ulcer. On the other hand it may be slight but persistent and prolonged and entirely unrecognized unless the stool is tested for occult blood. Thus, an unexplained anæmia may result so extreme at times as to resemble the primary form. Even more important is the tendency of polyps, which in the beginning are benign tumors, to undergo malignant degeneration, more often in the single but possible also in the multiple growths. Once this change has taken place, the clinical history gradually becomes that of cancer of the stomach.

Unless a polyp has become carcinoma and has developed into a tumor large enough to be palpated, nothing definite can be detected by *physical examination*. Ordinarily even extensive polyposis of the stomach does not produce a mass that can be felt through the abdominal wall.

Almost invariably in this disease *gastric analysis* fails to show any free HCl. Achlorhydria practically is constant. An abundance of mucus commonly is found in fasting contents and in all extractions after a test meal, and the material obtained is often colored by fresh blood. In a few instances the diagnosis has been made by finding fragments of a polyp in the contents removed from the stomach.

For years there was no evidence upon which dependence could be placed for the diagnosis of gastric polyps, and only at autopsy or operation was the condition ever recognized. Their presence in the stomach might be guessed at, even in patients with no previous symptoms at all or with, at most, vague and indefinite gastric discomfort and indigestion, when sudden hæmatemesis occurred or an obscure anæmia developed, and all

that was found was occult blood in the stools and achlorhydria with abnormal amounts of mucus in stomach contents. But there was no certainty of diagnosis until the *x-ray* technicians found a method by which polyps could be demonstrated during life, and a gastroscope was devised by which they could be brought directly into view. Carman in 1919 first revealed them by x-ray films in a stomach afterwards operated upon by Balfour, and Schindler in 1922 first described their appearance through the gastroscope. Carman, however, found only two cases of polyps in 50,000 x-ray examinations of stomachs and thought they must be very rare. Since his original report by the use of different and more perfect technique x-ray examination has constantly increased the number of cases recognized. The characteristic picture presented in the films is one of irregular, rounded, trans-radiant areas within the barium shadow, resembling the mottling of finger-prints, and multiple indentations or defects along one or both curvatures of the stomach. In the case of the large single polyp, it appears as an elongated tumor, cylindrical or club-shaped, with a broad base as a rule or else pedunculated.

Thus, it will be seen that, while polyps of the stomach may long be benign and symptomless, they may sooner or later form a mass large enough to interfere seriously with the stomach's motility or even block the pylorus with all the disturbance of function and production of pain that such interference causes. They may, at any time, bleed sufficiently to bring about extensive hæmatemesis and mælena, or else slowly and persistently until severe anæmia results. Furthermore, they are prone to undergo malignant degeneration and so to develop into a tumor that is nothing less than cancer of the stomach. For all these reasons the removal of gastric polyps should be

advised as soon as they are discovered, whether they have already led to complications or have so far been comparatively silent. Surgeons agree that the method of removal indicated is not simply excision but complete resection of the tumor-bearing portion of the stomach.

A man, aged 66, entered the Medical Clinic Ward of Stanford Hospital in September 1934. His story was that about three weeks before he began to have indigestion, characterized chiefly by gas and a feeling of distension after eating. More recently he began to vomit, from a few minutes to several hours after meals. Taking further food or soda would not relieve his distress. A few days before entering the Hospital, he had once noted a small amount of blood in his vomitus, not repeated. There had been no bloody or tarry stools. He had lost about ten pounds in weight since the onset of his illness. He recalled, when questioned, that for several years past he had experienced similar spells of indigestion with distress in the pit of the stomach and occasional vomiting but without blood or black stools.

On physical examination the man was well developed, slightly obese, extremely pale, looked weak and ill. Heart and lungs were negative, blood pressure 116 systolic, 70 diastolic. No abdominal mass was palpable, but there was distension by gas and increased tympany. Epigastric soreness was noted and increased resistance over the upper abdomen. The liver edge was palpable one cm. below the costal margin, moderately tender, smooth and firm. The tongue was not smooth. The scleræ showed no jaundice.

Blood examination showed hemoglobin 35 per cent., red blood cells 1,250,000, white blood cells 5,500, marked anisocytosis and poikilocytosis with macrocytes predominating. The icterus index was 9.4. Occult blood was mildly positive in the stool. The Wassermann reaction was negative. By gastric analysis achlorhydria was found, with low volumes, much mucus, old blood in fasting contents and fresh blood in all extractions after the test meal.

X-ray examination reported a large polypoid filling defect

occupying the entire middle third of the stomach, due to a tumor mass. The pyloric end was normal in appearance.

Following intensive therapy with parenteral solution of liver extract, the reticulocytes rose to 41 per cent. in five days with a subsequent rapid fall; after that the red cells gradually increased in number to 3,150,000 and hemoglobin to 62 per cent. Transfusion of 550 c.c. of blood was then given, followed by operation October 6th by Dr. Emil Holman. A large polypoid mass was found taking its origin from the lesser curvature and extending down over the posterior surface of the stomach, to which it was attached. There were no liver metastases. Gastrectomy of the middle third of the stomach was done by a sleeve resection. The polypoid mass after removal measured  $12 \times 9 \times 5$  cm., and microscopical examination proved it had become malignant. The patient made a rapid recovery, and when last observed, in March 1935, felt perfectly well. His blood count then was 3,500,000 red cells, with hemoglobin 84 per cent. Anisocytosis and poikilocytosis still persisted.

A similar case was reported in the Case Records of the Massachusetts General Hospital, in the New England Journal of Medicine, November 15th 1934. In this patient likewise the chief symptoms and signs were those of pernicious anemia with a typical blood picture and complete absence of free HCl in stomach contents. But the x-ray films showed an irregular filling defect, involving the lower half of the stomach, and gastroscopic examination revealed a rounded, elongated tumor with smooth contour, attached by a broad base. It resembled a benign polyp, but the broad base suggested malignancy. After preliminary treatment by liver extract and transfusion, operation was done. It showed a large polyp attached at the fundus of the stomach and running down its entire length, eventually passing through the pylorus into the duodenum. The polyp was removed but in this instance was not found

malignant. In the discussion that followed presentation of the case record the statement was made that about 40 per cent. of gastric polyps become malignant and at least 35 per cent. are associated with pernicious anemia. The frequency of the latter relation seems to be increasing.

## CHAPTER IX

### GASTROPTOSIS

Doubt is sometimes expressed as to whether a low position of the stomach in the abdomen has any disturbing influence upon digestion. This doubt arises because of the frequency with which x-ray examination discloses such a ptosis without apparent effect upon the patient's health. Nevertheless, case records accumulated throughout a number of years convince the writer that gastropptosis is associated many times with chronic disturbances of digestion and that correcting the former removes the latter. The reason that controversy has arisen about this question probably is that a broad enough view has not been taken. For gastropptosis usually forms only one part of a general visceroptosis with prolapse of all abdominal organs, particularly of the transverse colon as well as of the stomach and with this is associated neurasthenia, as described originally by Glenard in 1885. These cases are some of them congenital, some of them acquired. They occur in either sex but more often in women than in men, and they may appear at any age.

There is no typical clinical history about the character of the digestive disturbance to suggest that gastropptosis causes it. On the contrary, the vagueness, multiplicity and indefinite nature of the symptoms arouse suspicion rather than any fixed, routine complaints. There are other items in the story, however, that help to identify the cause of the long-continued disturbance of health. First, these patients usually are slender and poorly nourished and always have been. They complain

of inability to gain weight and are always substandard in nutrition when weight is compared with height. Second, they all complain of lack of vitality. So long as they can remember, they have always been delicate, unable to accomplish much, easily exhausted by effort, incapable of withstanding strain. Third, many of them suffer from backache when compelled to be much on their feet, that goes away when they are at rest in bed. Fourth, it is surprising how many of these patients give an account in their history of operations done by a surgeon for gastric ulcer or appendicitis or gallbladder disease to cure their stomach ailments, or by a gynecologist, in the case of women, for diseased ovaries or a retroverted uterus, to relieve their backache, or by an eye-specialist, who has fitted glasses because of the headaches, which so often constitute one symptom. Fifth, the medical practitioner often is equally to blame for treating these patients inadequately by diet and drugs for various disturbances of gastric and intestinal function, not recognizing the real underlying cause of all the symptoms.

On *physical examination* the picture presented by the typical visceroptotic is diagnostic. There is a small body, undernourished, with a long, lean, attenuated figure. The thorax is elongated and narrow, the costal angle high and acute. Characteristic findings in the abdomen are first, a prolapsed right kidney, sometimes merely the lower pole palpable, sometimes so far down it can be caught and retained after a deep breath, sometimes felt as a tumor floating free below the costal margin. The left kidney also may be prolapsed but far less often than the right. Second, the lower abdomen is more prominent than the upper, not only when the patient is standing erect but even when lying flat on the back. Third, in poorly nourished individuals with thin abdominal wall the outlines of the stomach

frequently can be seen, situated so the greater curvature lies some distance below the umbilicus, or if not visible ordinarily, it can be made so by inflating it with CO<sub>2</sub> by the method described in the diagnosis of pyloric obstruction. Fourth, in the women who have relaxed, flabby abdominal walls as a result of child-bearing, it is possible to observe irregularities of contour due to shifting patterns of intestinal coils. Men less often present the acquired type of visceroptosis, because there is no such cause operative as in women, where repeated pregnancies frequently over-stretch the abdominal wall, but congenital configuration of body such as described is frequently found in men with gastropptosis and general visceroptosis.

Commonly but not in every case of gastropptosis the stomach contents show complete and persistent *achlorhydria*. As a rule, secretion is deficient even if not entirely absent. This is the result of faulty position and innervation rather than of organic change in the stomach wall such as occurs in chronic gastritis or cancer or syphilis. But in some cases of gastropptosis there is found abundant mucus in stomach contents as well as deficient acid, indicating that a true chronic gastritis has ensued as a complication. In some instances also prolapse of the stomach is associated with increased rather than decreased secretion, so that the symptoms suggest gastric ulcer. That there is impairment of motility in these patients is proved by the faulty trituration of contents obtained after the Ewald test meal and by the fact that the stomach is not empty even after the various extractions made for analysis during one or two hours. There is certainly no routine finding by gastric analysis that affords conclusive proof of gastropptosis, but the evidences of impairment of both secretory and motor functions are the ones most often seen.

*X-ray examination* is of value in three different ways. First, it shows no defects in the gastric outline that would indicate ulcer or cancer as a cause of the patient's symptoms. Second, it proves that motility is impaired because the stomach empties slowly, and there is partial retention after six hours. Third, it demonstrates more accurately than any other method the existence and degree of the stomach's prolapse. There are differences of opinion about how low in the abdomen the stomach must lie before a diagnosis of gastropotosis can properly be made, but there seems to be agreement that when the lesser curvature is found below the level of the iliac crest with the patient standing erect, this constitutes an undoubted abnormality. Symptoms of disturbed digestion may be produced, however, by even lesser degrees of prolapse.

### DIAGNOSIS

The story of long-standing disturbances of digestion with symptoms often vague and indefinite of many kinds and not corresponding to any type of gastric disease with recurring bilious attacks and "sick headaches", with more or less constant backache, constipation and lack of energy, with slender body, poor nutrition and inability to put on weight, ought at least to suggest what is wrong. When to this history is added the evidence obtained by physical examination, the long slender body, the lack of adequate fat, the prolapsed right kidney, the prominent lower abdomen, the flabby abdominal wall, the impression previously created receives further confirmation. Add to this a gastric analysis showing anomalies of secretion and impairment of motility without any proof of intrinsic organic disease unless chronic gastritis has arisen as a complication and the final demonstration by x-ray films of a stomach

lying abnormally low in the abdomen or even in the pelvis and emptying slowly, then the original conjecture as to diagnosis becomes a certainty upon which adequate treatment can be based

Two dangers, however, confront the diagnostician in accepting gastropptosis as the cause of the patient's complaints. First, he is apt to forget that the abnormal location of the stomach, even though it gives rise to chronic disturbances of its secretory and motor functions, does not account for all the symptoms these patients present. This is only one part of a general visceroptosis involving all the organs in the abdomen and associated with a lack of nervous energy or neurasthenia. This conception makes intelligible symptoms such as constipation, backache, headache and an inability to cope with the routine duties of life, that can not be explained on the basis of gastropptosis alone. This also explains why direct treatment to the stomach by diet and medication never suffices by itself.

The second danger is that the physician, having satisfied himself that gastropptosis exists, may forget the possibility of complicating organic disease. Nothing about the position of the stomach in the abdomen makes it immune to cancer, for instance, nor does it prevent the addition of gallbladder or appendix disease to the clinical picture. If only the possibility of these or other complications is kept in mind and search is made for the symptoms and signs that identify them, they will be recognized without difficulty.

### TREATMENT

By non-surgical methods of management much good can be accomplished in these cases of gastropptosis. In fact it is not a condition calling for operation of any kind, for even if the

stomach is suspended, no benefit will follow, if the transverse colon is not likewise supported and the whole abdominal contents lifted. To afford such support, no surgical procedure has ever been devised that equals in efficiency a properly fitted corset for women or a belt for men. This is the first essential in treatment. The support should be worn constantly during the day. It should be adjusted in the morning before the patient gets out of bed and assumes the erect posture. With the body resting on shoulders and heels and the hips and abdomen elevated so that the viscera will gravitate toward the diaphragm, the corset or belt should be fastened snugly in place. At night, when the support is not worn, the foot of the bed should be elevated slightly, not to exceed four inches, so that gravity will aid in keeping the abdominal organs nearer their normal position. Finally to assist the stomach in getting the food started on its way through the pylorus, the patient should lie down on the right side for a half hour after each meal.

Important as are support and posture in the treatment of gastroptosis, almost equally important is diet. Apparently the factor most active in the production of digestive disturbance is atony or impairment of the stomach's motility. Secretion of acid may be increased or decreased, but if motility remains unimpaired, digestion proceeds fairly well. A soft, non-residue diet, such as that advised for chronic gastritis, is the one most often found suitable. Hyposecretion is the rule, but if gastric analysis shows hypersecretion, then the second list recommended for gastric ulcer should form the basis of the diet. Too much bulk at any one meal should be avoided, food should always be thoroughly masticated, and five small meals a day are often tolerated better than three large ones. No list can be given

that is absolutely correct for every patient, and addition or withdrawal of various articles can be made according to what seems to agree and what does not. In other words, each patient must be treated as an individual problem.

Treatment of gastropotosis by drugs is less important than the other measures so far described. A few drops of tincture of *nux vomica*, not to exceed ten, in a little water before each meal, to stimulate motility and some preparation of cascara at bedtime, if constipation is a troublesome feature, usually are the only drugs required. Medicines are of value in this condition to relieve symptoms, but there are none that can affect the underlying visceroptosis upon which the symptoms depend.

It is most important always to impress upon these patients that they must live within their capacity. Their bodies can not be reconstructed, and the model on which they are built never runs well at high speed. They must learn to recognize their limitations and keep within them, for their ambition almost invariably exceeds their capacity, and they are constantly attempting to undertake more than they can perform.

A woman, aged 46, had suffered from her digestion for 8 or 9 years. She had a good appetite, but sometimes right away, sometimes two or three hours after eating, she felt too full and became greatly distressed by a sense of distension. Sooner or later she began to belch and finally, after expelling much gas, obtained relief. Then she was comfortable until she ate again, but trouble recurred promptly as soon as she filled her stomach. Because of this sequence she had abstained from food and had practically starved herself, losing thus about fifty pounds since her illness began. Two capital operations had been performed to cure her, first removal of her gallbladder and several years later of her appendix, uterus, both tubes and ovaries. Numerous teeth also had been extracted without benefit, and her operations had made her no better, rather

worse. This woman was tall, slender, sallow, emaciated, and her figure was the typical one of visceroptosis. She had a prolapsed and palpable right kidney and a prominent lower abdomen. Gastric analysis showed an abundance of thick mucus and very deficient secretion of acid. X-ray films proved that the stomach was low in the abdomen with lesser curvature below the level of iliac crests, and that there was delay in emptying time. By means of a proper corset, rest after meals, a soft non-residue diet, tincture of nux vomica before each meal and cascara at bedtime, this patient after one month improved so much that she reported "I have no more gas, and everything I eat agrees with me". Seen one year later she still remained well and had gained twenty pounds in weight.

## CHAPTER X

### REFLEX DISTURBANCES OF GASTRIC FUNCTION

#### I. APPENDIX DYSPEPSIA; II. GALLBLADDER DYSPEPSIA

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Like Gaul in Caesar's time all disturbance of the stomach's functions is divided into three parts; that due to organic disease of the stomach itself, that due to organic disease elsewhere in the body, and that due to faulty innervation without organic disease discoverable anywhere. Not all chronic "stomach trouble" by any means is the consequence of gastritis or ulcer or cancer or any other demonstrable change in the organ's structure or position. In many cases extragastric disease, inside or outside the abdomen, reflexly interferes with the work the stomach is expected to do, and in a third group of patients, without any definite pathological lesion that can be located, exaltation or depression of nervous force may seriously interfere with the proper performance of gastric functions. In the reflex group most frequently perhaps the real disease is inside the abdomen, but it may at times be far removed and still cause disturbances of digestion; then the clue to what

the stomach symptoms mean may be found where least expected, in some organ not concerned with digestion at all. Hence the necessity for complete examination of the body and the realization that there are other parts besides the stomach and bowels that require consideration.

## I. APPENDIX DYSPEPSIA

### *Clinical History*

Chronic appendicitis is one of the chief extragastric causes of indigestion. By its presence it may disturb the function of gastric secretion, so that it is increased or decreased, and that of motility, so that it is stimulated or retarded. Hence there is no typical disorder of the stomach that points in every instance to the appendix as its cause. All sorts of manifestations may occur of greater or less importance, varying in constancy as well as in character and degree even in the same patient. A large group give a history resembling that of gastric ulcer, because hyperchlorhydria is a factor in each. These patients suffer more or less persistently from sour stomach. They complain about this for years, blaming their diet, taking soda for relief, with remissions for days, weeks or months, when there is little distress, followed by periods when annoyance is constant and severe. The similarity to ulcer may extend even so far as to include hæmatemesis as one of the manifestations. Finally there comes an attack of a different kind that calls attention directly to the appendix, or else without awaiting such an attack, the long-continued indigestion leads the patient to seek an investigation of its meaning. Then by various methods of examination it is discovered at last where the real trouble lies.

In another group of patients the symptoms suggest chronic gastritis. Everything they eat causes distress soon afterwards.

They are afraid to take this or that food and are constantly eliminating certain articles from their diet. They feel too full after eating little. They belch gas frequently. Some complain chiefly of distension, some of poor appetite, some of nausea. In this group as in the former the complaints are not constant. They may not appear at all for days. Then they recur without known cause and last for an indefinite period. The explanation of these cases usually is impaired gastric motility with atony. The proof that a definite relation exists between these disturbances of the stomach's functions and a chronic appendicitis is supplied by the frequency with which appendectomy, with release of chronic adhesions, results in complete removal of the indigestion. But in spite of every care a wrong conclusion now and then does occur, and removing a diseased appendix does not invariably remove the patient's complaints.

Besides the chronic stomach disorder, for which advice is sought, there are other incidents in the history that help to locate the cause. But these may make their appearance only at long intervals and thus be forgotten by the patient unless special inquiry is made about them. They concern disturbances of intestinal function rather than of gastric. Attacks of cramps with diarrhoea, appearing suddenly, commonly attributed to some food that has disagreed, lasting for one or for several days and subsiding after castor oil or some other cathartic but leaving the abdomen for some time afterward tender and sensitive, or occasional spells of several days duration, characterized by a sense of fulness and soreness in the right lower abdomen, a feeling of something there in the way, with discomfort increased by jarring such as results from an auto trip or riding horseback or dancing, or more severe attacks of pain associated with constipation, relieved after the bowel is emptied,

all of these incidents, with numerous variations, should be looked upon with suspicion. No doubt they would attract more attention unless they seemed to the patient less important than the chronic stomach trouble that constitutes the chief concern.

On *physical examination* no signs are found as a rule over the upper abdomen except moderate tenderness in spite of the distinctness with which the history points to the stomach as the organ involved. Over the lower abdomen and the right lower quadrant definite abnormalities may or may not be found. At times they are present, at times they are not, in the same patient. Tenderness on deep pressure, increased tension and muscle spasm and a palpable elongated mass that can be rolled under the examining fingers may be perfectly obvious at one examination, but at another time none of these signs may be discovered. This difference is due to the varying activity of the inflammatory process at one period as compared with another. The finding of such abnormalities as those mentioned is always significant, but the failure to find them at any one examination does not exclude chronic appendicitis. Even though no such signs are ever found, extensive pathological changes may nevertheless have taken place in and about the appendix, sufficient to cause all the reflex gastric symptoms. No other form of chronic appendicitis so frequently causes dyspepsia as that called obliterative, which has buried the appendix under a mass of adhesions and caused it to atrophy. Yet this pathological change affords little or no evidence of its presence on physical examination. The retrocecal appendix likewise, adherent to the posterior wall of the bowel, is commonly quite inaccessible to palpation. For all these reasons, when signs are found, they add much to the understanding of

the diagnostic problem, but when they are not, their absence does not prove that chronic appendicitis is not a factor in the production of the patient's complaints.

As regards *gastric analysis* the most common finding in reflex appendix dyspepsia is hypersecretion, but any sort of secretory curve may be observed, above or below normal or within normal limits. There is no secretory abnormality that is diagnostic. The presence of vegetable husks or fibres or other food fragments in the fasting over-night contents and poor trituration after an Ewald test meal indicate impaired motility. But there is no retention of consequence, no mucus or blood is found, and the stomach is clean.

The evidence that *x-ray examination* supplies is of two kinds, eliminative as regards the stomach and demonstrative as regards the cæcum and appendix. In the first place it proves that no organic change has occurred in the stomach, no matter what the history suggests, and in the second place it frequently reveals abnormalities in and about the cæcum that are highly significant. These comprise delay in the terminal ileum, fixation of the cæcum, tenderness over its inner side, cæcal stasis and delay in its emptying time. The appendix itself may or may not be visualized. If visible, it may be fixed and tender, unusual in shape and size, kinked or angulated or clubbed or beaded and much longer than the average. It is frequently slow in emptying, retaining barium for forty-eight or even seventy-two hours. If these signs are found, they are definite additions to the proof that the patient has a chronic appendicitis. If the appendix is not visible, the absence of these signs does not prove that it is normal, for it may lie retrocæcal and thus be obscured by opaque material in the cæcum, or its lumen may be obstructed so that the barium can

not enter to make it visualized, or it may be obliterated, atrophic and buried by adhesions.

### *Diagnosis*

Reviewing the facts upon which this depends, there is a history of chronic disturbance of gastric function, but of many kinds, with no type diagnostic. The history includes not only symptoms of stomach derangement but also those attributable to disturbance of bowel function, such as persistent constipation, occasional intercurrent attacks of diarrhoea with cramps and more or less constant soreness, tenderness and dull pain over the right lower abdominal quadrant. From the history alone no conclusion can be reached, but a suggestion at least is given. Physical examination reveals signs that are often vague and indefinite and may be overlooked at one examination though obvious at another, or may never be discovered at all. Gastric analysis is variable in its results, inconclusive and never diagnostic, but it is of value because it aids in excluding organic gastric disease. X-ray examination likewise is of greatest value in eliminating ulcer, cancer and other intragastric disease that might cause the symptoms. It may also demonstrate beyond question that the appendix is abnormal, or it may give no help at all in proving where the pathological change lies that causes the patient's ill-health. It is only by combining the various items of evidence, acquired in all the ways described, that a trustworthy conclusion can ultimately be attained. Even so, in any obscure case, where symptoms are not urgent and delay does not imperil life, it is better to proceed slowly before taking action and to await developments while the patient is kept under observation.

It can not be too strongly impressed that chronic disturb-

ances of gastric function are not always due to chronic appendicitis even when it has been proved that the two co-exist. It is important, therefore, to be reasonably certain about the relation between them before advising appendectomy. Too many times the appendix is removed without benefit to the patient's health because some other cause was really responsible, undiscovered in advance of operation.

### *Treatment*

A positive conviction that chronic appendicitis is the true cause of the patient's digestive troubles justifies advice that the appendix should be removed. But occasional disappointment about results, when the same symptoms persisted even after the appendix was out, has led the writer to avoid haste in such cases and to employ six months or even a year in observing the patient before urging resort to surgery. During this time the diet is arranged to suit the stomach's functions as shown by gastric analysis. Medication is prescribed to neutralize hyperchlorhydria or else to stimulate secretion and motility as indicated. The bowels if constipated are kept free by a mild laxative such as cascara. Furthermore during this period of "watchful waiting" investigations are repeated from time to time, not only of the gastrointestinal tract but also of other organs that might reflexly cause the symptoms presented. In this way the ultimate decision that appendectomy is required results more frequently in success, and sometimes on the other hand such delay convinces the diagnostician that appendectomy is not indicated and would not benefit.

1. A man, aged 37, complained that for years past many foods upset him completely, such as meat, raw fruit, various desserts,

not only causing distress in his stomach but a feeling of congestion in his head. His bowels were obstinately constipated. Physical examination was entirely negative. Gastric analysis showed a normal secretion. X-ray examination demonstrated no abnormality in stomach or duodenum, but the appendix was shown to be fixed and immovable and remained filled by the barium for forty-eight hours. Treatment for nine months by diet and medication resulted in no improvement, so that finally appendectomy was advised. This revealed that the appendix and the head of the colon were bound down by old and very dense adhesions, that the appendix was sharply angulated at its base close to the bowel and its distal portion distended and thickened. Following removal of the appendix and release of all adhesions, the patient's symptoms all disappeared. Nearly twenty years have elapsed since that operation, and the symptoms have never recurred.

2. A second patient, aged 42, for five or six years had had stomach trouble diagnosed as duodenal ulcer, with recurring attacks of pain, belching, waterbrash, burning, with nausea and vomiting, appearing one hour or two after meals, lasting for weeks at a time, and the symptoms never entirely disappearing even on treatment for ulcer. At the time he sought advice he had no such pain, nausea or vomiting, but complained of a heavy, dragging feeling in the pit of his stomach at all times after eating. No signs were found on physical examination over the upper abdomen, but decided tenderness and a palpable finger-shaped mass over the appendix area. Gastric analysis showed a high-grade hyperchlorhydria with total acidity 84 and free HCl 72. By x-ray films no defect was noted in stomach or duodenum, but the cecum was fixed, the appendix indistinctly outlined, tender to pressure. As the patient was no better after six months observation and medical treatment, operation was advised. When the abdomen was opened, a fibrous band was found extending from the base of the cecum to the abdominal wall, the cecum was twisted upon its axis and bound down to the wall of the pelvis by old inflammatory adhesions, the appendix was buried behind the cecum, bound down by adhesions there. Since

this operation, freeing all restricting bands and removing the appendix, five years have elapsed, and the patient has never had any further trouble.

## II. GALLBLADDER DYSPEPSIA

### *Clinical History*

Next in frequency to chronic appendicitis and perhaps equaling or exceeding it as a cause of interference with gastric functions comes *chronic cholecystitis*. It does not always disturb digestion, but it does so often enough to make it imperative to investigate the possibility of gallbladder disease in every case of chronic dyspepsia. Sometimes the gastric symptoms simply form a background for other manifestations that point directly to the gallbladder itself. Sometimes they comprise the only evidence of disease over long periods of time. Sometimes they do not occur at all, even when attacks of gallbladder inflammation recur frequently and are unmistakable. Thus it becomes possible to formulate groups of cases of chronic cholecystitis with reference to the effect they produce upon gastric functions. The first of these presents a history of recurring attacks of biliary colic with good health and digestion between attacks and no complaint whatever of the stomach. The second group relates a story not only of these recurring attacks of severe pain but also of constant stomach trouble between them. In the third group the chronic disturbance of digestion becomes the chief complaint with no violent pain at any time but only minor intercurrent attacks that implicate the gallbladder, such as discomfort experienced from time to time in the right side below the ribs, a feeling of fullness and soreness or a dull ache and sense of distension. These symptoms may annoy for days at a time, then disappear entirely for weeks or for months.

In the fourth group the stomach symptoms are practically the only ones about which complaint is made, and though active gallbladder attacks have subsided, reflex indigestion persists. The character of the gastric disturbance varies. Most often it corresponds to that seen in chronic gastritis. There is complaint of discomfort soon after eating, repeated eructations of gas, poor appetite, occasional nausea, but particularly prominent is the story about "gas". Sometimes, but less frequently, the symptoms suggest gastric ulcer, because sour stomach appears late after meals with heartburn, waterbrash, nausea and at times vomiting of sour fluid. In exceptional instances, associated with weight loss and anorexia the history may lead to a suspicion of gastric cancer.

When reflex dyspepsia is due to gallbladder disease, the stomach on physical examination gives no signs. There is no tenderness over it, no tumor, no visible peristaltic wave. Furthermore physical examination supplies little evidence that the gallbladder itself is diseased. All that is found may be entirely subjective, not objective, but in some cases the signs may be perfectly plain and definitely objective. That the findings are negative so often is not surprising, when it is recalled that the gallbladder lies normally beneath the costal margin, under the liver edge, entirely inaccessible by palpation through the abdominal wall. Furthermore as a consequence of chronic cholecystitis the gallbladder frequently becomes a small, contracted, shrunken organ, still more difficult to palpate. Therefore, even though the changes produced by chronic inflammation are causing reflexly constant disturbance of gastric functions, nothing abnormal can be detected by external examination. Tenderness, no matter by what method or maneuver elicited, never by itself justifies the conclusion that the

gallbladder is diseased, because some other organ in the same area may really be the sensitive one. The most that can be expected in chronic cholecystitis on physical examination is increased resistance and rigidity in the right hypochondrium, and even this may be inconstant, present at one examination and not at another.

There seems a general agreement about the frequency with which gastric hyposecretion accompanies chronic cholecystitis, but not about the exact degree of this frequency. In the writer's experience gastric analysis shows complete achlorhydria in approximately 25 per cent. of these patients and deficient secretion, below the normal average, in another 25 per cent. In other words, in at least half the cases of chronic cholecystitis, gastric secretion is absent or decreased. In the other half, however, secretion is normal or may be definitely above normal. In the stomachs with hyposecretion an abundance of mucus also is found commonly, so that the condition corresponds in these cases to that of chronic gastritis. Then it is probable that the symptoms of which complaint is made are not due simply to reflex disturbance of secretion but to the chronic gastritis. Therefore, in any case of chronic dyspepsia, when gastric analysis shows chronic gastritis and when no other cause for the latter can be found, disease of the gallbladder should be suspected. But when stomach contents show only disturbed secretion, either above or below normal, without the presence of mucus in excess, it becomes probable that the disturbance of secretion and the symptoms of dyspepsia which this causes are reflex in origin.

*X-ray examination* shows no important abnormality in the stomach itself in this group of cases where gallbladder disease disturbs the stomach's functions. The organ may be atonic,

it may empty slowly, it may have flabby walls and be dilated, but it shows no defect in its contour, and it empties in six hours. As regards the gallbladder direct examination of it by x-ray is extremely unreliable, and films must not be expected to show changes in its size or the density of its walls that are sufficiently definite to be accepted as proof of pathology. Even gallstones do not appear in the films unless they are calcareous, and this is the case in not more than twenty-five per cent. Indirect evidences of gallbladder disease shown by x-ray, due to the effects of adhesions around the organ, such as flattening or deformity of the duodenal cap, abnormalities in the course of the duodenum beyond the cap, reverse peristalsis in it, or a high fixed position of the hepatic flexure of the colon, are also unreliable aids in diagnosis. There may never have been any pericholecystitis to produce these abnormalities, or if they are shown, they may have been caused by disease outside the gallbladder and so may not rightly implicate that organ.

At the present time the x-ray evidence that is most reliable regarding the gallbladder is that obtained after the patient has received the dye, tetra-iodo-phenophthalein, either by mouth or by vein. This dye is removed from the blood by the liver and passes with the bile into the gallbladder. After hours have elapsed, fifteen as an average, the dye becomes concentrated so that the gallbladder is shown much more distinctly in x-ray films than it ever is without the dye. By this method of examination, introduced by Graham and known as cholecystography, valuable evidence is obtained. If the gallbladder is normal and performs its function of concentration as it should, a shadow of its outline is produced that indicates exactly its size, site and shape. If the gallbladder is diseased and has

lost its power of concentration, the shadow is faint and indistinct and may not appear at all. Non-calcareous stones, not demonstrated by a flat plate at all, may now be seen as negative shadows, that is, the space they occupy can not be filled by the concentrated dye, and they show themselves as lighter spots against the opaque background of the rest of the gallbladder contents. Valuable as this new method of diagnosis has become, like all others, it is not infallible when taken alone, and the evidence it supplies should be considered only in connection with all the rest obtained in every possible way.

### *Diagnosis*

Given the problem to determine the cause of a patient's chronic disturbance of gastric functions, what facts prove that this is chronic cholecystitis? Summing up the evidence obtained by the different methods of investigation described, the symptoms most often resemble those found in chronic gastritis but may resemble those of ulcer instead. Usually there are other symptoms referable to the gallbladder itself in intercurrent attacks of greater or less severity. No physical signs over either stomach or gallbladder are found constantly, nor are any diagnostic. Gastric analysis may show the presence in the stomach of mucus in excess with absent or deficient secretion of acid, so that chronic gastritis associated with chronic cholecystitis, rather than reflex disturbance alone, is the cause of the symptoms. But when hyperchlorhydria is found, the symptoms must be interpreted as the result of reflex disturbance. Finally, x-ray examination proves there is no organic change in the stomach, duodenum, appendix or colon to explain the clinical history and may show definite signs of disease in the gallbladder. By combining the evidence obtained by all

these methods and not trusting to any one alone, reasonable certainty as to diagnosis as a rule can be attained.

But opportunity for error is always present, and in spite of every possible care in collecting and weighing evidence the outcome sometimes proves the conclusion reached was wrong. There are several ways in which this comes about, and these can be better illustrated than described. First, the symptoms and signs may resemble gastric ulcer so closely that this is accepted as the pathology causing the chronic gastric disturbance.

A woman, aged 50, complained of annoyance by gas and sour stomach for twenty years; she had bloating and distress after every meal; her appetite was good, but everything she ate seemed to sour and turn to gas; there was much belching and frequent sour eructations; occasional sick headaches with profuse vomiting; pain in the stomach and through to the back, coming on at variable intervals after food. For months past she had regularly induced vomiting or else washed her stomach out to secure relief. She was well-nourished, rather fat than thin; the stomach was dilated, prolapsed, atonic and splashing. No abnormality was found elsewhere in the abdomen. After a test meal the total acidity was 96 and the free HCl 69. Blood was present both in stomach contents and feces. In spite of negative x-ray report regarding the stomach, operation was recommended for ulcer. None was found, but the gallbladder was thickened, adherent to the duodenum, enormously dilated, contained an ounce of muddy bile, one large gallstone and eighteen smaller ones. Following its removal all symptoms disappeared.

Second, one of the most difficult tasks is to decide when the gastric symptoms are caused by chronic cholecystitis and not chronic appendicitis. Sometimes the picture presented is very deceiving.

A woman, aged 44, complained of stomach trouble of nine months' duration, characterized by poor appetite, a sense of fulness and pressure after food, gradual loss of weight and strength, but no actual pain in the stomach, no nausea, no vomiting. For five years past she had suffered more or less constantly from a sore spot and a darting pain in her right side just at the lower border of the ribs. Nine months previously at the outset of her present dyspepsia she had an acute attack of pain in this spot, followed by jaundice for several weeks, and since then she had never been well. While under observation in hospital she had a similar attack of pain at the right costal margin with a sense of soreness and tightness there. She was obese, showed decided rigidity and tenderness over the gallbladder area but nowhere else in the abdomen. The stomach was dilated and gastric analysis showed a hyperchlorhydria with total acidity 70 and free HCl 50. No defect in gastric contour was shown by x-ray. At operation no disease in stomach or gallbladder was found but an old chronic appendicitis with many adhesions.

Third, the patient's history may closely resemble that of gastric cancer, when there is no disease in the stomach at all, and the symptoms are all reflex from gallbladder pathology.

A man, aged 53, always previously well, had been ailing about three months. He complained of poor appetite, a sense of fulness and oppression soon after eating, much belching of gas and loss of weight amounting to twenty pounds since his illness began. Abdominal examination was negative, not only as regards the stomach but also the gallbladder. Gastric analysis showed much thickropy mucus, retained food particles in fasting contents, with both free and occult blood and complete absence of free HCl after a test meal. No defect in the stomach's contour was shown by x-ray examination. The patient did not improve on diet and medication, but all his symptoms persisted, and he continued to lose steadily in weight, strength and color. Finally exploratory operation was advised because cancer of the stomach was suspected. This showed

no abnormality whatever in the stomach but a large single gallstone completely filling the gallbladder, measuring  $3 \times 2$  cm. After cholecystectomy his symptoms all disappeared, and he gradually regained his lost weight.

All the preceding case histories were obtained before cholecystography had been made available as a means of diagnosis. Since that time it has been possible to reach more accurate conclusions. The following case demonstrates the value of this comparatively recent method of investigation.

An obese Italian, aged 40, complained of stomach trouble for five months, constantly, without intermission, characterized by poor appetite, much belching of gas, no vomiting but frequent nausea. He had no pain after eating, but about two hours after a meal he felt weak, perspired and became nervous and faint. Since his illness began, he had lost 35 pounds in weight, although he was still obese. This stomach trouble all started after a sudden severe attack of pain in the upper abdomen, running through to the back, lasting all one night and followed by jaundice for two or three days. He had never been well since but had experienced no further attack of that kind. Physical examination of the abdomen was negative. Gastric analysis showed a secretion within normal limits. X-ray films taken after the dye demonstrated very incomplete filling and poor concentration of a gallbladder of normal size, containing one indistinct ring-shaped shadow. Medical treatment for six months did not benefit, so operation was advised. The gallbladder was found filled with stones, with its walls greatly thickened and many adhesions about it. Following its removal, complete recovery resulted.

### *Treatment*

During the last few years the trend has been distinctly toward more conservative treatment of chronic cholecystitis. In the absence of severe pain, particularly of typical attacks of

biliary colic, with no proof that the gallbladder contains stones, where the diagnosis depends mainly upon reflex indigestion, with faulty filling and emptying of the gallbladder shown by cholecystography, Graham warns that the results of cholecystectomy are likely to be unsatisfactory in approximately 40 per cent. of the cases. It is no longer in order, therefore, to advise surgery as soon as the diagnosis is made, but rather to employ medical treatment at the outset until its efficacy has been tested.

The first object of such non-operative treatment of chronic cholecystitis is to remove so far as possible all foci of infection from mouth, teeth, tonsils and sinuses, in order to minimize the chances of re-infection of the biliary tract. The second object is to overcome stasis and promote free biliary drainage. This is accomplished (a) by a diet that stimulates the gallbladder to contract and empty, containing particularly such fats as egg-yolk, butter, cream and olive oil, with meats in moderation and acid fruits, but avoiding all highly seasoned, rich and excessively coarse foods, and all alcoholic drinks, that may produce or aggravate a coincident chronic gastritis and duodenitis; (b) by daily moderate exercise, such as walking by preference, or by horse-back riding, golf, tennis, rowing or bowling; (c) by drugs that stimulate the output of bile from the liver, particularly the bile salts, in a capsule or pill at bedtime, containing bile salts one grain (0.06 gm.), extract of cascara and phenolphthalein of each a half grain (0.03 gm.), of which the dose is one or two; (d) by drugs that prevent stasis in the territory drained by the portal vein, particularly the colon, such as a mixture of magnesium sulphate and sodium sulphate of each one part with sodium phosphate two parts, of which the dose is one or two heaping teaspoonfuls in hot water in the

morning before breakfast. If no benefit follows such a plan of therapy within at most three months, cholecystectomy should be advised. But not infrequently by such measures patients lose not only their chronic disturbance of digestion but also all other evidences of gallbladder disease.

## CHAPTER XI

### DISTURBANCES OF GASTRIC FUNCTIONS WITHOUT PATHOLOGY: GASTRIC NEUROSES

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This constitutes the third group of cases with disturbance of gastric functions, but this time without organic disease discoverable in the stomach or anywhere else in the body. In this group the disturbances are apparently due to faulty innervation and are known as “gastric neuroses”. Much doubt has been cast upon the existence of such cases, the argument being that pathological changes somewhere in the body cause the reflex interference with digestion and that the failure to find it does not prove there is none. But when no evidence can be found of organic disease, that might explain the symptoms of indigestion, the only diagnosis left is gastric neurosis. Formerly this group was believed to be a large one, but little by little it has been contracted. Certainly with increasing experience fewer cases are given this designation, and less satisfaction is felt in leaving them with it. Improved methods of investigation in

recent years and persistent observation of patients for the discovery of new manifestations or developments of their ailment not infrequently lead to the shifting of cases from this diagnostic group to some other as time goes by.

A woman, aged 52, complained of difficulty in swallowing, for which no explanation could be found except cardiospasm after careful and complete investigation. After six months treatment by diet, antispasmodic medication and bougies without cure, exploratory laparotomy revealed two large stones in the gall-bladder, mulberry in type. After cholecystectomy the cardiospasm never returned.

### I. DISTURBANCES OF MOTILITY

The two gastric functions of most importance are motility and secretion, and practically all cases of disturbed function, however produced, concern one or the other of these two. Thus, when the cause is faulty innervation, the manifestations may affect mainly motility or mainly secretion or may involve both.

(1) When by decrease in the supply of nerve force motility is impaired, the condition that results is classified as *atony of the stomach*. Its characteristic features are early satiety at the table, ability to eat little before feeling too full, persistent flatulence and belching and more or less sense of distension after meals. On physical examination the most significant sign is a succussion splash hours after food has been taken. The fasting stomach usually is empty, but after a test meal, particularly after the Ewald which requires trituration, there is slow emptying, and amounts above the average are extracted by the stomach tube. These contents usually show on analysis secretion within normal limits with no excess of mucus, no occult blood and no other evidence of organic disease. X-ray films

demonstrate that the stomach's contour is normal, and though the stomach is slow to empty, it does not retain food. Organic pyloric obstruction thus is eliminated. If gastropptosis is shown by the films, atony is commonly associated with it, because of the coincident neurasthenia that forms a part of the clinical picture.

This lack of normal power in the stomach wall is essentially one manifestation of nervous exhaustion and is a common consequence of the wear and tear of daily life. It follows, therefore, that the first essential in treatment is to secure for the patient more rest, to stop the expenditure of undue energy and to limit activity within reasonable bounds. Diet should be so arranged that food will give the weakened stomach as little work to do as possible. Soft diet should be advised, such as recommended in the list for chronic gastritis. Bulk at each meal should be avoided and particularly fluids with the food should be restricted. If three regular meals of this character fail to maintain the patient's weight, two smaller ones may be added, one during the forenoon and one during the afternoon. The only drug that seems to be of benefit in these cases is *nux vomica* or strychnine, ten drops of the tincture or one fiftieth grain (1.25 mgm.) of the alkaloid before each of the three main meals. Occasionally atony involves the intestinal as well as the gastric wall, so that cascara in the form of the fluid or dry extract is needed to overcome constipation.

(2) When the patient suffers from increased rather than decreased gastric motility because of over-stimulation, one or the other of several remarkable manifestations may appear. There are a number of different forms that hypermotility may take, but the most striking ones are aerophagia, nervous vomiting and rumination.

(a) Aerophagia is the name given to attacks of persistent, noisy belching without relation to meals or to the kind or amount of food eaten. These eructations are so loud, so explosive and repeated so frequently without apparent cause that usually it is possible to recognize their nature at once, even before the patient is examined. In this condition the individual swallows air unconsciously and then brings it up again voluntarily. There is no odor to the air thus expelled, as there is so often to the gas produced by the improper digestion of food. Absolutely no sign of organic disease in the stomach is found to explain the symptom. The excuse offered by the patient for the act is that the stomach feels too full and belching gives relief. This form of gastric neurosis may persist for days or weeks at a time, particularly under the influence of any excitement or nervous strain. If sufficient time is taken to examine carefully the entire body, and if gastric analysis and x-ray films of the stomach are made a part of the investigation, the patient sometimes acquires enough confidence in the physician to accept his conclusion that the belching is entirely unnecessary. The writer in a few instances has seen positive assurance to this effect suffice to end aerophagia at once. But psychotherapy alone does not always succeed, and then dependence must be placed on nerve sedatives such as bromides and belladonna as well as upon psychotherapy, by suggestion, reassurance and adjustment of patient to environment. Sometimes also the administration of drugs such as asafoetida or valerian, that make the eructations offensive to the patient, will cause their discontinuance.

A woman, aged 32, complained that for several weeks she had stomach trouble characterized by loud, noisy expulsions of gas in rapid succession, whenever she was nervous or excited. This

came on as the result of shock following the death of her father. She never had the trouble at night, had a good appetite and had lost no weight. The patient was a large, well-developed woman, and no evidence of organic disease was found on physical examination. Gastric analysis showed normal secretion. Her trouble disappeared promptly after a few doses of a bromide and valerian mixture.

A man, aged 38, made his diagnosis known while waiting in the reception room before he entered the private office by violent, noisy paroxysms of belching. He said these were not constant but recurred whenever he became excited or nervous. He had a good appetite, never vomited, had maintained his weight constantly. No evidence was found of any organic disease, but corneal and pharyngeal reflex were absent. He belched noisily, violently and continuously during the course of his physical examination. Gastric secretory curve was slightly below the average normal; urine and blood were normal; Wassermann reaction was negative. X-ray examination showed "no radiographic evidence of an organic lesion of the gastrointestinal tract; there was normal filling and emptying of a normal sized gallbladder which reacted well to the fatty meal". This man improved on anti-spasmodic medication, particularly belladonna. But now and then paroxysms would still recur when he became excited.

(b) Nervous vomiting is a troublesome condition to treat because it occurs usually in nervous, excitable women, who present numerous other signs of irritability and instability, in consequence of which they are unreasonable and disinclined to accept advice or direction. The story is of repeated vomiting, occurring as a rule soon after a meal, without nausea preceding, without effort and no matter what food has been taken. The amount of the vomitus usually is not large. This may take place after every meal day after day, and yet a singular feature of the situation is that in spite of all this loss of food the pa-

tient does not lose weight or does so very gradually. There is no evidence found by physical examination, by gastric analysis or by x-ray investigation that any organic disease of the stomach or of any abdominal organ is responsible for the persistent vomiting. But there are other disturbances noted in the history or by physical examination that suggest a disordered nervous system. Furthermore there are almost always factors in the patient's life and environment that cause restlessness and excitability, and it is particularly when something has happened to irritate or annoy or to produce unusual fatigue or emotional strain that vomiting begins which may persist for days afterward. There is no specific treatment by drugs for this manifestation of gastric hypermotility. The object is to break up the bad habit acquired, and to accomplish this is almost impossible so long as the patient remains in the usual environment. Hospital management offers the best prospect for cure, by isolation, rest, frequent feedings by mouth supplemented if necessary by subcutaneous infusions of normal salt solution, intravenous injections of glucose solution and rectal feedings to which bromide and chloral may be added. The psychological effect of such a plan is as important as the physical.

A woman, aged 37, gave a history of vomiting after every meal constantly for two years and at intervals for two years before that. She had good appetite and enjoyed her food, but she felt full and oppressed shortly after eating, and without nausea she simply regurgitated what she had eaten. This relieved her distress immediately. She was a large, well-nourished woman, rather plethoric than anemic, vivacious, loquacious with no appearance of ill-health. No evidence was found of any organic disease on physical examination. Gastric analysis showed normal secretion. By x-ray examination the stomach was proved to be normal in size and position, there was no

evidence of pyloric obstruction, no delay in emptying time, no defect in contour. A diagnosis of nervous vomiting was made, based upon the fact that there was no relation between the rejection of food and the quality or quantity taken, the good state of nutrition, the occurrence of vomiting so soon after eating without preliminary nausea, the ease with which it was accomplished and the absence of any evidence of organic gastric disease. Her husband stated that at home she cried frequently about unimportant matters and at times would scream at the top of her voice. She refused to go to a hospital. No treatment recommended had any apparent effect. When last heard from, she was still vomiting but without any deterioration of her general health.

(c) *Rumination*. — This is a rare disorder in which the patient habitually and voluntarily regurgitates into his mouth the food from the stomach at a variable time after swallowing it, not to eject it but to swallow it again with or without further mastication. It is essentially a bad habit, not due to organic disease, entirely a neurosis, with no serious results to the patient's health. The writer has the record of one such case in a Japanese, who was observed at intervals over a period of twenty years and never ceased his habit during all that time. He died of hemachromatosis with ascites, which developed late in the course of his rumination. No advice and no treatment seemed to have any effect in overcoming the patient's peculiar conduct with regard to his food. He appeared to enjoy tasting it twice.

## II. DISTURBANCES OF SECRETION

These are much more difficult to explain on the basis of faulty innervation alone. They are found so frequently as a manifestation of organic gastric disease or of disease elsewhere in the body reflexly disturbing secretion, that the discovery of

wide deviation from the normal always implies that some other explanation exists than that of gastric neurosis, and this explanation usually can be found, if careful enough search is made.

(1) *Achlorhydria* means the absence of free HCl from not only the fasting stomach contents but also from all extractions after a test meal, its absence not only after an ordinary Ewald or alcoholic test meal but also after a subcutaneous injection of histamine with its persistent absence after such stimulation and not for the first hour only. Confusion arises now and then as to the meaning of the terms achlorhydria and achylia. They are not synonymous, for the former means the entire lack of free hydrochloric acid in stomach contents, while the latter properly means complete absence of all gastric secretion, not only of acid but also of the ferments pepsin and rennin as well. Commonly, however, the names are used interchangeably, because the absence of acid is considered the essential abnormality, and because there are no simple routine tests to determine the presence or absence of the ferments.

So many diseases are known to present achlorhydria as one manifestation, that it is difficult to believe it ever constitutes a disease by itself. It may, however, appear at times to be purely functional with no pathology discoverable in any organ to account for it after prolonged observation and careful search. Sometimes an underlying general neurosis seems responsible for it, such as neuræsthenia, hysteria or melancholia, depressing all nervous activity, but in other patients perfect health is maintained, and no complaint is made of any kind, in spite of persistent achlorhydria or so-called "achylia gastrica".

However always before achlorhydria is assumed to be purely functional, all other possible causes must be reviewed, such as

are found in gastric and extragastric disease. (a) In the first group probably the most common one is *chronic gastritis*, easily recognized by gastric analysis, by x-ray films and by gastroscopy. *Cancer of the stomach* ultimately produces achlorhydria in many cases but not in all and never until late in the course of any case. Too much emphasis has been placed in the past upon this sign in the diagnosis of gastric cancer. X-ray films of the stomach furnish much more reliable proof. In *syphilis of the stomach* absence of acid is the finding usually described, but before it is accepted as the cause there should be found also a positive blood reaction, an x-ray film showing extensive defect in stomach contour, response to specific therapy and possibly histological examination of tissue removed at operation. It is well to recall the fact that this is one of the rarest forms of gastric disease. In *gastroptosis* achlorhydria may be found persistently with or without other evidence of chronic gastritis. Here it is probably a true neurosis of secretion due to associated neurasthenia. Proof that the patient has gastroptosis is supplied by physical examination and by x-ray films, but it is probable that depression of nervous energy has more to do with the abnormal secretion than has the low position of the stomach in the abdomen. Finally with *gastric polyps* almost invariably achlorhydria has been found associated. X-ray films and gastroscope will tell whether this is the cause.

(b) Among the second group of extragastric diseases producing achlorhydria or connected with it, the most interesting instance is *pernicious anæmia*. The association of these two is now generally accepted as essential, so that no case that shows free HCl in the gastric contents can be properly diagnosed as pernicious anæmia. Blood counts and smears, however apparently characteristic, are looked upon with suspicion unless

achlorhydria also is found. *Combined sclerosis of the spinal cord* in practically every case shows persistent achlorhydria. Disturbances of sensation and motion with gradually increasing anemia make the diagnostic picture, but achlorhydria may precede any definite manifestations of spinal cord disease, just as it may be found before the blood picture is typical in pernicious anæmia. There is undoubtedly some relation between *chronic cholecystitis* and achlorhydria, but in some of the cases, where the two are associated, the explanation is found in a complicating chronic gastritis. When there is no apparent reason for the absence of free HCl from stomach contents, investigation of possible causes should always include cholecystography and all other methods available for the recognition of gallbladder disease. The tropical disease, *sprue*, seen also at times in temperate climates, frequently but not invariably includes achlorhydria among its features. Sore tongue, diarrhoea and severe anæmia help in its identification. *Pellagra* is another cause of achlorhydria. Along with changes in the skin and the central nervous system it may produce numerous disturbances of digestion, and when the patient seeks advice about the latter, investigation commonly shows absence of free HCl. Finding the other features mentioned as well as the absence of gastric secretion should prevent error in diagnosis.

*Cancer of the digestive tract* beyond the stomach sooner or later gives rise to achlorhydria. This is true whether the site of the neoplasm is liver, pancreas, large intestine or even the rectum. This fact is explained partly by inhibition of the gastric glands by toxins produced by the growth, partly by progressive anæmia or, it may be frankly admitted, by some other factor not well understood. Other symptoms and signs indicate where the cancer is situated, and x-ray films prove that the

stomach itself is not responsible and may aid also in locating the real site of the disease. *Chronic portal hepatitis*, commonly known as cirrhosis of the liver, by obstructing portal circulation leads gradually to gastric stasis. Gastric analysis then frequently shows achlorhydria but usually associated with excess of mucus as well, so that the real cause of the disturbed secretion is a chronic gastritis. The clinical history and the signs found by physical examination commonly point to the liver as the site of the pathology underlying the digestive disturbance and the achlorhydria. *Myocardial weakness* with a failing heart now and then presents itself first in the guise of chronic indigestion, and when gastric analysis is done, achlorhydria is found. This is the result of faulty circulation through the stomach wall and inadequate blood supply to the secreting glands there. But other evidences of heart weakness can be found, when search is made for them, and the disturbance of gastric secretion becomes only one part of the clinical picture. In *myxædema* achlorhydria is found not infrequently. When secondary anemia is discovered also, as so frequently occurs in this disease, the combination may arouse suspicion of pernicious anæmia. Careful examination of the blood by count and smears, together with a test of the basal metabolic rate, usually will make the diagnosis clear. Finally, long-existing *pulmonary tuberculosis* or *chronic nephritis*, or any other depleting disease causing secondary anæmia and depression of nervous energy, may so interfere with gastric secretion that achlorhydria results. The discovery of this abnormality in the course of any such chronic disease should never be interpreted to mean that some complicating affection of the stomach has developed.

From the foregoing review it becomes evident how many conditions there are in which achlorhydria may be found with-

out assuming that a neurosis is responsible, and how careful the physician must be to make a complete investigation before accepting the conclusion that absence of free HCl is functional only without any underlying pathology.

What shall be done when achlorhydria is discovered? Whether treatment for it is indicated or not depends upon what symptoms it produces, or whether it produces any symptoms at all. First, if digestion is more or less constantly disturbed, and if an adequate explanation for the absence of free HCl is found in some organic disease of the stomach itself or of some other organ to which the achlorhydria is secondary by reflex action, then the measures required for the management of the primary disease may suffice to remove the effects of the disturbance of secretion. Second, if no adequate cause for the achlorhydria can be found, but nevertheless the patient complains constantly about distress after food, loss of appetite, belching and perhaps a chronic diarrhoea, then the condition should receive attention for itself, regardless of its cause. Third, if there are no symptoms whatever of disturbed digestion, no treatment is indicated even though by gastric analysis, in the course of a routine investigation, complete absence of free HCl is discovered. The specific remedy, when one is required, is dilute hydrochloric acid. The average dose is twenty drops, well diluted, after each meal with orange juice added to flavor, if the patient prefers. This dose may be repeated one hour after the first if indigestion persists, and again a third time one hour after the second, if needed to overcome the symptoms. This repetition only imitates the constant secretion usually poured into the stomach in health while digestion is going on.

(2) *Hyperchlorhydria*. — This disturbance of secretion likewise occurs so often in association with and apparently as a conse-

quence of gastric or extragastric disease, that great hesitation should be felt about assuming that it is ever purely functional. Not so many diseases seem to be responsible for increased as for decreased secretion. Prominent among those related to it are gastric and duodenal ulcer, chronic appendicitis and chronic gallbladder disease. These are common causes of reflex stimulation of the gastric glands to oversecretion and are the ones to be remembered as the most probable explanation when hyperchlorhydria is found. But when no evidence can be elicited that these organic diseases are present, and when no other can be discovered, then the only conclusion possible is that the hyperacidity is functional and due to nervous excitability. Hyperchlorhydria and hypersecretion are not synonymous terms, for by the former is meant that the amount of free HCl in the gastric secretion is above normal, while the latter indicates that the total volume of secretion is increased. The two are often associated but not necessarily so, and each is the result of overstimulation of the gastric glands either reflexly by disease inside or outside the stomach, or else without any discoverable disease at all.

Undoubtedly nervous and emotional states may effect the gastric secretion, such as prolonged worry, anxiety, grief and mental overwork, and these may suffice to account for hyperchlorhydria without any organic disease, constituting a true gastric neurosis. But, when by gastric analysis hyperchlorhydria is found, and this is associated with the symptoms usually attributed to it, such as heartburn, sour stomach and particularly the eructation during digestion of sour fluid that irritates the throat and sets the teeth on edge, suspicion should always be aroused that some other cause exists besides nervous disturbance. These patients should be kept under observation,

and developments should be carefully noted. The probabilities are always that some day the diagnosis of gastric neurosis will be exchanged for another that is more definite.

A man, aged 23, first seen in 1912, complained of stomach trouble for a year previous, characterized by heartburn, nausea about an hour after eating but no vomiting, no waterbrash, no pain, no loss of weight. No abnormality was found on physical examination, but an Ewald test meal showed moderate hyperchlorhydria only, and the case was listed as a gastric neurosis. This man was not seen again until 1933. He then reported that trouble of the same sort had recurred at intervals ever since, but in recent attacks pain had been an added feature, coming on about three hours after eating and relieved by food. Physical examination still showed no evidence of organic disease and gastric analysis a secretion within normal limits. X-ray films, however, demonstrated a clover-leaf deformity of the duodenal cap, so that the diagnosis was changed from gastric neurosis to chronic duodenal ulcer.

The acceptance of hyperchlorhydria as a satisfactory diagnosis by itself was much more common twenty or twenty-five years ago than now. At present it is looked upon only as one symptom of some disease that may not be obvious at the moment but will probably become so ultimately. There may be such an entity as excess of acid secretion in the stomach due only to nervous influences and properly called a gastric neurosis, but this diagnosis should be accepted with a mental reservation, and the patient watched subsequently to see what changes, if any, may occur. Meantime a diet should be advised such as that for ulcer; the excessive acidity should be counteracted by alkaline powders and belladonna, and the nervous perturbances by a bromide mixture or phenobarbital.



PART II

DISEASES OF THE INTESTINES



## PART II

### DISEASES OF THE INTESTINES

#### INTRODUCTION

Complaints about disturbance of intestinal functions are not so common as those about gastric. This is due partly to the fact that intestinal disease is less frequent and partly to the tendency of the stomach to speak for other diseased organs besides itself and for the intestines among the rest. Not infrequently when the symptoms are referred largely or entirely to the stomach, the real pathology is in the bowel.

Much information that is new has been acquired about intestinal disease in the past few years, and it is no longer so much of a "terra incognita" as it used to be. Not only have new explanations been found for long-recognized but not well understood forms of pathological change, but new groups have been segregated from old ones, so that intestinal disease has thus acquired a new interest for the practitioner during the past decade.

# CHAPTER XII

## METHODS OF INVESTIGATION

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The means available to determine the presence and nature of intestinal disease comprise history, physical examination, stool examination, various other laboratory tests, proctoscopy and x-ray examination.

### CLINICAL HISTORY

In addition to obtaining the usual routine account of how the patient's other systems do their work, as well as of the environment, occupation and daily habits of life, it is particularly important in the group of cases now under consideration to find out about how the intestines perform their functions. Certain pertinent questions are in order in every case. Do the bowels move regularly every day? If so, how many times? What is the character of the stools as regards consistence, form, color and quantity? Is a laxative taken, and if so, which one? Does pain precede or accompany defecation? Is there much gas passed with the bowel movement or at other times? In addition to such questions as these, which apply to all patients,

there are a number of other details about which information is desired in each special case under investigation according to the nature of the intestinal disease. These will be considered as each disease is discussed.

### PHYSICAL EXAMINATION

Too much should not be expected from this in the investigation of disease of the intestines. In a number of forms of serious disturbance of bowel function, no definite objective evidence can be found. In the groups characterized by chronic inflammation or even by ulceration of the bowel, for instance, no signs may be discovered except the subjective one of tenderness. It is only when the whole wall becomes involved and not simply the lining membrane that objective abnormalities are observed, or when special areas undergo pathological change, as in appendicitis, diverticulitis or regional ileitis, or when new growths involve the bowel wall. Interference with the normal contour of the abdomen always is significant. By inspection, distension may be noted, general or local. By palpation, abnormal masses may be found that were not visible through the wall, or an enlarged abdomen that is symmetrical throughout may be recognized as filled with fluid or with gas, or a flat abdomen that is rigid and board-like in consistence may be accepted as meaning a serious departure from the normal. By percussion additional information is obtained about the cause of abdominal enlargement, and by auscultation frequently it is possible to determine whether peristalsis is continuing properly or has ceased because the intestinal wall has lost its normal power. But in general the signs obtained by physical examination are less informative than those acquired by other methods of investigation. Never should be forgotten

in any ailment affecting particularly intestinal function the procedure called by Osler "the first duty of a consultant", namely, a digital exploration by rectum. Sometimes an explanation for symptoms can be found in this way when none whatever can be discovered by examining through the abdominal wall.

### STOOL EXAMINATION

As gastric analysis constitutes an indispensable method of investigation in disease of the stomach, so does stool examination in disease of the intestine. The physician by direct inspection can obtain much information of importance, even before the laboratory technician has carried out more or less complicated tests. The stool ordinarily is solid or semi-solid, rounded and firm, brown or yellow-brown and shows no extraneous substances such as mucus or blood. If the specimen submitted for examination is unusually soft or mushy or even liquid, it means haste through the colon so that time was not allowed for proper absorption and condensation of its contents. A cathartic always does this, but if none has been taken, it means abnormal function. On the other hand, fæces unusually hard and dry, or passed in lumps like sheep's excrement, indicate long delay in the bowel such as occurs in chronic constipation. Changes in the usual color of the stool frequently are due to some food eaten, such as spinach, which always makes them dark green or almost black, or blackberries, blueberries or huckleberries which likewise darken their color, or beets which make them red. In any case where some abnormal or unexpected color is noted, questioning the patient about diet often will afford the explanation. Certain drugs also will discolor the fæces and may cause needless alarm unless the patient is

warned. Preparations of iron will make the bowel discharges black and so will bismuth. Here again inquiry as to what medication the patient may have been taking, will sometimes clear up all doubts. It is well understood that putty colored, gray or white stools mean deficient bile in the bowel, and that black, tar-like stools mean blood poured out high up in the intestinal tract and acted upon by secretions it meets there before it is finally discharged. As regards extraneous substances in the stool, a moderate amount of mucus or so-called "slime" coating the stool or passed separately, while it is abnormal, does not necessarily mean disease, unless it is persistent and becomes excessive. It is only when the entire discharge is mushy and mixed thoroughly with grayish-white masses of sticky mucus, or when long shreds and flakes are found, that the condition becomes significant. Bright red blood in the stool most often means hæmorrhoids or a fissure of the anus, but may come from ulcer or cancer higher in the rectum. The symptoms associated with its passage help in locating its origin, and proctoscopic examination settles the matter. Even round worms, pin worms or segments of tape-worm can be easily recognized by gross inspection of the stool, though many times the patient suspects the presence of these parasites and brings the stool for proof, when the objects seen prove to be nothing but portions of food refuse or shreds or strings of mucus.

In the laboratory routine examination of the stool should begin with a chemical test for occult blood by the same method as advised for stomach contents. Then a small portion of the stool, the size of a bean, or about a teaspoonful of liquid or semi-solid material, should be mixed with 10 c.c. of distilled water and centrifuged until the supernatant liquid is clear. A portion of the fresh sediment is then dropped on a slide and examined

with the microscope for food fragments, blood and pus cells, ova and parasites. Finally a similar preparation is stained by the addition of a drop of Lugol's solution and again examined under the microscope. These simple methods suffice as a routine for office examinations. If tests of the bacteriology of the stools by cultures are required, these demand more elaborate and extensive methods of investigation and are best referred to a special laboratory such as is found in every well-equipped hospital.

### LABORATORY EXAMINATION

Laboratory examinations naturally should not be limited to the stool in the effort to secure a proper understanding of the character and the meaning of intestinal disease. The urine, the blood count and the Wassermann reaction should all be investigated as a routine in every case, and the information, thus acquired, may supply a clue to diagnosis that otherwise would be missed. Furthermore gastric analysis after a test meal is of almost as much importance as stool examination when the bowel is functioning abnormally. Finally, a test of the basic metabolic rate may furnish the explanation why diarrhoea persists, after all other methods of investigation fail.

### PROCTOSCOPY

In the recognition of ulcers and the characteristics of different varieties, in the discovery of tumors, in identifying the site from which bleeding proceeds, the proctoscope and sigmoidoscope have long been employed in the investigation of disease of the bowel. Valuable as these instruments are for diagnosis, like the gastroscope, they should not be used indis-

criminally but only by those who have had special training in their management. Much damage can be done by them if improperly handled, and much information can be overlooked or misinterpreted. Furthermore, even in the best of hands they do not permit very deep inspection of the bowel, and the lesion causing symptoms may lie beyond the point to which the instrument reaches. While this method of investigation has great value, its uses are limited, and the information it supplies needs to be supplemented by that obtained in every other way.

### X-RAY EXAMINATION

After barium is given by mouth, the stomach and duodenum are well visualized in x-ray films, and at the other end of the gastrointestinal tract, the colon from the ileocecal junction, particularly the cæcum itself, the ascending and transverse portions, usually are shown satisfactorily. But there is no proper visualization as a rule of the small intestine, and so approximately twenty feet of bowel are lost to observation, except in its terminal portion; and the lower part of the large intestine frequently is not shown at all or is much lacking in detail, because the barium has not passed far enough down to fill it or else has been discharged too soon from the rectum. No way has yet been found to visualize satisfactorily the small intestine throughout its course, but after a barium enema the large bowel can always be shown by x-ray films in every detail from cæcum to rectum. Barium by mouth is contraindicated, if there is suspicion of obstruction of the colon by a neoplasm or by any other lesion, because of the possibility that it may not pass, but barium by enema is always safe. Taking into consideration the fact that x-ray examination

may fail to give the information desired, and on the other hand that objections sometimes arise to its employment at all, it becomes evident that the usefulness of this method of investigation is more or less limited in intestinal disease. Nevertheless the assistance it does give is indispensable.

CHAPTER XIII

ACUTE INFLAMMATION OF THE INTESTINES

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This is a common ailment of humanity and most people experience it at some time or other. It is known by different names. By the laity it is usually called by the symptom that chiefly characterizes it, “diarrhoea”. By the physician it is designated, according to the part that seems most involved, acute enteritis, acute colitis or acute enterocolitis. It is not always possible, however, to decide to which portion of the intestinal tract the inflammation is limited, and probably there is never any sharp demarcation anyway between the point where inflammatory changes cease and normal bowel begins. It is more likely that in every case the acute inflammation is widespread but with its intensity greater in one section of the tract than another, thus determining what symptoms predominate in each case. After all, the means adopted to give relief are much the same, no matter in what part the inflammation seems most severe; so that diagnosis of the exact anatomical site involved becomes of less consequence.

CLINICAL HISTORY

The first and most characteristic symptom that marks the onset of acute intestinal inflammation is change in the fre-

quency and character of the bowel movements. Instead of the usual one daily evacuation at a regular time there are several passages at irregular times, and instead of the customary formed stool, the discharges become more or less soft or even liquid and watery. They frequently contain much mucus or so-called slime, sometimes mixed with a little blood, but with no such amounts or constancy of bloody content as is seen in other forms of acute inflammation where ulceration constitutes a part of the pathology.

With this change in the frequency and character of the stools comes pain. Beginning as discomfort, uneasiness, a sense of pressure and heaviness in the abdomen and consciousness of peristaltic activity, these abnormal sensations soon increase to active griping pain with a sensation of twisting and rolling of the bowels and cramps preceding the frequent movements. The manifestations of acute intestinal inflammation vary in number and degree, but diarrhœa with pain, soreness and cramps in the abdomen are the characteristic features. Bowel movements may be few or many, large or small in amount. They are practically always loose and thin. Pain may be experienced only preceding a movement or persist with greater or less intensity between them. In some cases, where the lower colon is chiefly involved, there may be an almost constant desire to defecate, known as tenesmus, with the passage of only small amounts of material that does not give relief. Fever usually is slight and brief in duration. If it is high and persistent, it calls attention to a greater than average degree of inflammation and should warn the physician that the symptoms may mean more than at first appeared. The course and duration of an ordinary attack occupies only two or three days to one week. But it takes some time after the

acute symptoms have subsided for the bowel to recover its normal functions, and relapse is frequent unless precautions are taken to prevent.

Most of the causes that lead to acute intestinal inflammation enter the bowel through the stomach. It is not surprising, therefore, that disturbances of that organ frequently precede or accompany those lower down. An attack often begins with nausea and vomiting, and epigastric distress, loss of appetite and discomfort after eating commonly are associated with the bowel symptoms. The causes usually operative are improper food and drink. Questioning the patient about the diet preceding an attack thus becomes an aid to diagnosis. Some sort of irritant is directly responsible. This may be mechanical, such as results when the patient has eaten too freely of corn, berries or other coarse foods containing much residue, or toxic, as when some food has been taken that is not fresh and has undergone chemical change, such as happens at times with fruit, cheese, fish or meat, or bacterial, when micro-organisms too numerous for the natural defenses of the body to overcome have been taken into the digestive tract with the food. Any case that at the outset seems to belong in the group now under consideration may ultimately prove to be due to some specific organism such as the *Entamæba histolytica*, the Shiga bacillus or the typhoid bacillus. In many cases, however, the exact micro-organism responsible can not be determined either by the microscope or by cultures or by agglutination tests. It is unwise, therefore, to postpone exact diagnosis and the treatment dependent upon it until the causative agent has been identified.

## EXAMINATION

*Physical examination* does not afford much information except that there is generalized tenderness, moderate distension by gas, and more or less rigidity due to protective muscle spasm, but there is no localized tenderness as in appendicitis and no palpable mass. The absence of such signs helps in eliminating other diseases, but the signs that are found are often indefinite.

*Stools.* — Direct inspection of the bowel discharges tells much about the nature of the patient's illness, even without the aid of the microscope and the culture tube. They are always abnormal in consistence, soft, unformed, sometimes entirely liquid, and frequently show mucus of viscid, gelatinous consistence, in long strings or in clumps. Blood mixed with the mucus or passed by itself is not seen except in the ulcerative types of the disease. Frequently the stools are foul in odor, particularly at the outset of an attack. Sometimes bubbles of gas are interspersed. The color most often seen is light yellow, but it may be green or gray. The microscope and cultures can not tell much more, except that in persistent, virulent cases and those where blood is definitely and constantly present in the stools, these more scientific methods of examination may reveal the Shiga bacillus or the *Entamæba histolytica* or the typhoid bacillus. In the ordinary attack the patient is better or practically well before such laboratory reports are received.

## DIAGNOSIS

Sudden onset of illness with or without a history of some preceding indiscretion in diet; abdominal discomfort, soreness,

pain and sense of peristaltic unrest; increased frequency and abnormal character of bowel discharges; slight fever or none at all; the whole attack not exceeding one week in duration; these are the features that combine to make the diagnosis of ordinary acute inflammation of the bowels. But this picture may vary, and the variations observed should not cause confusion. Many times an attack begins with vomiting, and this symptom persists for hours before intestinal disturbance occurs. The greatest distress may be experienced either in the upper or the lower abdomen. Violent cramps may precede each bowel movement or not occur at all. Tenesmus, the almost constant desire for bowel evacuation with straining effort and small amounts expelled, is a symptom only when the inflammation is most intense in the descending colon, the sigmoid and the rectum. This symptom with stools consisting almost entirely of gelatinous mucus possibly stained by blood characterizes the form of bowel inflammation known as dysentery. The passing of pure blood in considerable amount may possibly occur in an acute ileocolitis of severe degree with intense congestion of the mucous membrane, but it always suggests ulceration due to some specific cause such as the Shiga bacillus or *Entamæba histolytica*.

All this sounds simple and unmistakable, but there are a number of opportunities for serious error. The first of these is *acute appendicitis*, never to be forgotten as a possibility in any acute attack involving the abdomen. This disease may make its onset with vomiting followed by diarrhoea, and there is no way in the beginning to distinguish this localized form of inflammation from the more general one. But even though pain, tenderness and increased resistance to pressure at first are widespread over the abdomen, they gradually become limited to the right

lower quadrant. The diarrhoea is less frequent in appendicitis and, if it occurs, is less persistent. There may be none after the first few movements. Fever rises gradually, also the leucocyte count. There is ultimately, after a few hours, a greater degree of fulness, tenderness and rigidity found in the right lower abdomen than anywhere else. Sudden cessation of the pain, followed by comparative comfort, should not delude, for this change does not occur naturally in acute intestinal inflammation and not in acute appendicitis either except when a swollen appendix ruptures. It is therefore a bad sign rather than a good one.

When the *Entamæba histolytica* invades the bowel, the consequence is so-called *amæbic dysentery*. This appears in a sudden attack characterized by severe abdominal pain, nausea, and vomiting, intense desire to defecate, tenesmus, with stools at first semi-solid, then liquid, then consisting largely of bloody mucus in small amounts. As a rule there is no fever, but in severe attacks the temperature may reach 101° or 102° F. The severity of the suffering, the passage of blood, the intensity of the paroxysm should arouse suspicion, but stool examination is required for positive diagnosis with the finding of the parasites there.

The same sort of an acute attack may be caused by the Shiga bacillus giving rise to *bacillary dysentery*. This makes its onset like the former with sudden and violent pain in the abdomen, frequent loose bowel movements containing blood, tenesmus, tender abdomen, fever and rapid pulse. The toxæmia this organism produces is greater, the fever higher, the prostration more extreme and the illness in general more serious. There is no way, however, by history or by clinical course to distinguish this with certainty from other severe forms of

acute intestinal inflammation. Microscopical and cultural examinations of the stool are essential to make the diagnosis.

Other diseases commonly beginning with diarrhoea or presenting it as the first manifestation of sufficient importance to attract attention are typhoid fever, pernicious anæmia and hyperthyroidism. In *typhoid fever* diarrhoea usually is an early symptom but preceded for a few days by such symptoms as anorexia, headache and lack of energy. There is no such violent or explosive onset as characterizes amœbic or bacillary dysentery, and there may not even be any pain associated with the frequent bowel movements such as is seen in an ordinary attack of acute intestinal inflammation. Sometimes, however, there is enough pain and tenderness in the right lower quadrant to suggest acute appendicitis, and laparotomy has been performed for this, and the true condition recognized only after the abdomen was opened, so close is the resemblance in some cases. The persistence of the diarrhoea, the gradual step-like rise of temperature, the discovery of rose spots, positive blood cultures or Widal reaction, with normal or low white count in spite of increasing fever, finally make the diagnosis plain.

Mild forms of acute diarrhoea without pain or fever may occur in the course of *pernicious anæmia*, recognized by blood counts and smears or in *hyperthyroidism* even without the other symptoms and signs that are typical but with increased basal metabolic rate. Rarely such diarrhoea may be due to faulty elimination of poisons from the blood by the kidneys in *chronic nephritis* and Nature's effort to expel them through the bowel. There is no need for error in recognizing any of these conditions in which diarrhoea plays a part, if care enough is taken to make every investigation possible by history, physical examination and laboratory tests.

## TREATMENT

Assuming that it has been determined that the disturbances of bowel function are really due to acute inflammation of its lining wall and are not the expression of some other underlying disease of more serious import, the plan of treatment to be adopted is well defined.

First, the patient should be put at rest in bed. This may not be absolutely essential in all cases, but it shortens the course of the disease and hastens recovery. The patient can be assured that by this method a few days will accomplish more than ten days or two weeks of attempts to cure without rest.

Second, as soon as possible one ounce (30 c.c.) of castor oil should be given, but only provided the physician is certain that the condition is not due to acute appendicitis. This old-fashioned remedy not only clears the bowel of the offending material that is causing the inflammation but has a subsequent sedative effect upon the intestine. If coincident vomiting makes it impossible for the patient to retain castor oil, then there should be substituted a course of calomel and soda powders, one quarter grain (15 mgm.) of the former and one grain (60 mgm.) of the latter every half hour until twelve such powders are taken. These not only have an ultimate purgative effect upon the bowel but an immediate sedative effect upon the stomach. If the bowels do not move freely in response to these powders, then the liquor magnesiæ citratis of the pharmacopeia (purgative lemonade) should be given in two ounce (60 c.c.) doses each hour, beginning one hour after the last powder, until the desired effect is produced; or if the stomach will not tolerate this remedy, a soap-suds enema should follow the calomel and soda powders, or an enema consisting of Epsom salts two ounces

(60 c.c.), glycerine two ounces (60 c.c.) and water to make one pint (500 c.c.).

Third, until this initial stage of clearing out the bowel has been accomplished, no thought should be given to diet. But after the first twelve hours of treatment usually it is proper to begin liquid food. This should be as nearly as possible free from micro-organisms; therefore weak broths and cereal gruels should be preferred. Milk is not a good food for these cases because it always contains many germs, even the certified varying from the other varieties in this regard only by having its number of bacteria restricted to a lower figure than theirs. For the first day or two patients do better with but little food, so that the inflamed intestine may have rest. Four ounces (120 c.c.) every two hours may be given at the outset, of any clear broth, beef, mutton or chicken, cooked with rice or pearl barley, then strained clear, and seasoned with a little salt but no pepper. To alternate with this broth, any cereal gruel may be given in similar amount, made from farina or any other wheat food, from rice or from barley, or from Eskay's food or Nestle's food or imperial granum. Any of these gruels may be seasoned with a little salt. Still another permissible food is malted milk, made by adding the powder to hot water according to the directions that the package gives. After the pain, diarrhoea and tenderness have subsided, as they usually do in two or three days, toast, rice and soft cooked eggs may be added to the diet, but meats, vegetables and fruits should be resumed very slowly. A bowel once acutely inflamed regains its normal power only gradually; it remains irritable and easily upset; so that too rapid a return to ordinary diet is apt to retard recovery. It is usually at least two weeks after the simplest attack before the patient can expect to eat again as he did before it occurred.

Fourth, after the bowels have been evacuated thoroughly by castor oil or by calomel, it is in order to restrict further peristaltic activity. For this purpose the most useful combination is one of opium and bismuth. The following prescription has frequently been used with satisfaction in such cases as these now under consideration; tincture of opium five minims (0.3 c.c.), bismuth subcarbonate twenty grains (1.3 gm.), in mucilage of acacia, syrup of tolu and anise water to make two drachms (8 c.c.). This dose is to be given every two hours at first, but only in case cramps and diarrhoea persist. If after the first dose pain and diarrhoea recur before the second is due, it is given when the two hours have elapsed; but if they have not recurred, the dose is not repeated until they do, no matter if three or four hours pass by between times. Thus automatically the administration of opium is regulated, and it is not continued more frequently than the need for it exists. Usually after the first two or three doses at two hour intervals, the time between them becomes longer and longer, and after pain and diarrhoea cease entirely, no further medicine is given. Thus an obstinate constipation is avoided as a sequel of the attack.

Fifth, at the outset, persistent vomiting, interfering with the administration of drugs or of food, as well as causing the patient much distress, often may be relieved quickly by gastric lavage with hot bicarbonate of soda solution, one teaspoonful to the pint. When no food can be taken because it causes nausea, albumen water, made by stirring the white of one egg into an ordinary drinking glass of cold water, often can be retained in tablespoonful doses. Incessant desire for bowel evacuation, with only a little clear or bloody mucus passed each time, is relieved best by washing out the lower bowel with warm normal salt solution, and then injecting opium into the rectum, using ten

to twenty minims (0.6 to 1.3 c.c.) of the tincture in one or two ounces (30 to 60 c.c.) of starch water; or by a rectal suppository containing one grain (0.6 gm.) of the aqueous extract of opium and one-eighth of a grain (7 mgm.) of extract of belladonna in cocoa butter; or if these fail, then by hypodermatic injection of morphine sulphate one-quarter grain (15 mgm.) with atropine sulphate one one-hundred-and-fiftieth grain (0.4 mgm.). It is also useful to recall that in patients showing great weakness and prostration, the camphorated tincture of opium (paregoric) is more stimulating than the plain tincture and can be substituted in dose of one or two drachms (4 to 8 c.c.) every two hours for the opium and bismuth mixture, used with the same precautions about its repetition.

CHAPTER XIV

CHRONIC INFLAMMATION OF THE INTESTINE

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Classification of diseases of the bowel, where chronic diarrhœa is the characteristic feature, has always been confusing. Naming the disease anatomically is practically impossible because of the difficulty in deciding in each case just what portion of the intestine is chiefly involved. Naming it according to the etiological factor that produced it is a little more satisfactory but not entirely so, because there are so many cases where this factor is unknown and can not be identified. It is generally accepted, however, that the symptoms observed are almost invariably due to disturbance of the functions of the colon rather than of the small bowel, for about the latter and its

diseases not much is known. Thus it becomes proper as well as convenient to classify all the cases presenting chronic diarrhoea as their chief symptom by the general term, chronic colitis. Next it is noteworthy that in this large group there are some patients that habitually pass variable amounts of mucus with the bowel discharges but no blood, while others present the constant or frequent addition of blood. On this basis it becomes possible to divide chronic colitis into two subdivisions known as non-ulcerative and ulcerative with the dividing line between the two the presence of blood in the stools.

I. The non-ulcerative subdivision is observed clinically in two forms; (1) a mild, painless variety with more or less constant passage of mucus in moderate amounts and, therefore, designated catarrhal, and (2) a more severe variety, characterized by recurring attacks of colic with the passage of large amounts of mucus in strips or sheets or even casts of the bowel, known clinically as mucous colitis. II. The ulcerative subdivision is also split up into four parts according to etiology as determined in the laboratory. These are named (1) amoebic, (2) bacillary and (3) tuberculous colitis, with (4) still another group that has to be designated non-specific ulcerative colitis because its exact etiology is still undetermined but with clinical features that distinguish it from the other varieties named. This classification has proved a practical one clinically and will be followed in this consideration of diseases of the intestine.

#### NON-ULCERATIVE GROUP

##### *Chronic Catarrhal Colitis*

This occurs more commonly than any other form of colitis and may persist for months or years without causing much impairment of health or any severe suffering. It is due usually to the

same causes as chronic gastritis and is often associated with it. These causes include first of all habitual indiscretion in the taking of food and drink, too much food or food of improper quality, too highly seasoned, poorly masticated and triturated, taken at irregular hours or too many times a day. Second, the cause may be chronic stasis of the circulation through the bowel wall, due to portal obstruction or to a weak myocardium. Third, the habitual use of purgative drugs taken without proper advice and control, used too frequently or in too large a dose over long periods of time, not infrequently is responsible for chronic catarrhal colitis.

*Clinical History.* — All functions of the bowel are disturbed by this disease, motility, secretion and sensation. First, the characteristic feature is too many evacuations of the bowel, or what is commonly known as chronic diarrhœa. Instead of one stool each day or possibly two, several occur during the twenty-four hours. These may all be passed during the early morning or the forenoon, or they may annoy the patient at any and all times of the day and night. This irritability of the bowel and intolerance of its contents may be replaced for a few days by the opposite condition of impaired reaction to stimulation and constipation. Then the former diarrhœa is resumed, and from time to time these two abnormal states alternate. Such disturbances of motility are common to all forms of chronic colitis and not peculiar to this one only.

Second, the character of the bowel movements is abnormal. Partly due to hasty peristalsis and hurry through the colon, but partly also to increased secretion of mucus by the bowel's lining membrane, the stools are, most of the time, not formed and solid as they ought to be, but mushy or semisolid or even liquid in consistence. Mixed with them is an unusual amount

of mucus, making the material glairy and sticky and visible as gelatinous clumps, or strings or shreds. Even when the stool is formed, mucus may be seen coating it, white or grayish in color and giving a smooth appearance as if the material had been varnished. This habitual passage of mucus in greater or less amount each day is the most characteristic feature of chronic catarrhal colitis. The consistence of the stool and the number of evacuations may vary from time to time but the mucus persists.

Third, there are abnormal sensations in the abdomen produced by this disease, not so much pain in the form of cramps and colic as a feeling of distension, pressure and discomfort with rumbling and noisy gurgling. This is experienced particularly in the early morning, frequently disturbing sleep then and persisting until the bowel is cleared of its irritating contents and of accumulated gas by the discharges that take place after rising. The rest of the day may be fairly comfortable in an average case, but may be disturbed off and on in another patient by recurring defecations. Reflex disturbances of the stomach such as flatulence and frequent belching of gas may add to the patient's discomfort at all times.

Fourth, general health may be impaired by long continued chronic catarrhal colitis. Patients who suffer from it are apt gradually to lose weight and to develop a sense of weakness and inefficiency. They tire easily and lack energy for persistent effort. Ultimately they undergo also a change in disposition, become irritable, fussy, despondent and given to introspection.

*Physical examination* adds little to the diagnostic evidence. No very definite signs are found. The most significant one is tenderness along the course of the colon, more decided sometimes over one part than over another but usually pretty well

distributed along the ascending, transverse and descending portions of the large bowel. This sign, however, is entirely subjective. Objective signs are not found except that occasionally the whole abdomen is slightly swollen and resistant as well as tender. This absence of objective evidence, however, is of value because it excludes localized disease such as chronic appendicitis, diverticulitis, tuberculosis and neoplasm, that might be responsible for the symptoms observed.

*Laboratory Examinations.* — (a) It is not always possible for the physician to have at his disposal the aid of a skilled laboratory technician to make special investigation of the *stools* for ova and parasites and for tests by cultures, but it is always possible for him to make direct inspection of the bowel discharges as to their general character and the presence or absence of mucus and blood. Not only the recognition of chronic catarrhal colitis but also its differentiation from the other more severe forms, especially the ulcerative, may be accomplished by gross inspection of the stools.

(b) *Gastric analysis* is of importance in every case of intestinal disease, because chronic gastritis often accompanies the bowel inflammation and lack of proper secretion, and the finding of achlorhydria may explain the chronic diarrhoea. In other instances, even though the pathology is in the colon and nowhere else, reflex gastric symptoms may cause more complaint than the real disease, and by gastric analysis it is possible to prove that the stomach is not the organ to which treatment should be directed.

(c) *Blood studies* made as a routine will call attention to the cases of diarrhoea which are due to pernicious anemia. (d) Tests of the *basal metabolic rate* will explain an occasional diarrhoea due to hyperthyroidism. (e) *Urinalysis* will give the clue to a

diarrhoea compensatory in character from uræmia when kidneys fail to eliminate properly. The stools alone may suffice to make the diagnosis of chronic catarrhal colitis, but these other laboratory tests may point the way to proper therapy.

*X-ray Examinations.* — Like physical examination the value of this method of investigation in chronic catarrhal colitis is greater for what it eliminates than for what it demonstrates. There may be observed greater haste than normal about the passage of the barium meal through different parts of the colon; there may be a smeared appearance of the bowel wall due to the presence of mucus; there may be an absence of normal haustrations in the transverse and descending colon; while these signs are of value when taken in connection with other evidence, they are not so constant or so definite that they become diagnostic when taken alone. But it is by excluding the changes indicative of chronic appendicitis, or of disease in the terminal ileum or the ileocæcal region, tumors in the wall of the colon or diverticulitis, that x-ray examination is chiefly of value in the diagnosis of chronic catarrhal colitis.

*Diagnosis.* — A long continued history of bowel movements abnormal as regards their number and character with more or less constant discomfort and uneasiness in the abdomen; the finding on physical examination of tenderness along the course of the colon but no other evidence of organic disease; the observation on gross inspection that the stools are mushy or semi-solid or even at times liquid with the visible addition of mucus; an x-ray report of hasty peristalsis, a smeared appearance of the bowel lining and a smooth contour of the colon instead of the normal haustrations; these are the features that combine to make the diagnosis of chronic catarrhal colitis practically certain. The absence of any history of violent

attacks of colic accompanied by the passage of large amounts of membranous or stringy mucus excludes the variety known as mucous colitis, and the absence of severe pain and tenderness as a more or less constant feature with the presence of blood frequently in the bowel discharges and a less decided interference with general health prove that the colitis is not one of the ulcerative types.

But chronic diarrhoea may be only one symptom of changes in the course of the bowel more serious than those of simple catarrhal inflammation. Underlying and producing the abnormal condition may be *chronic appendicitis*. But then the colitis appears only in recurring attacks with comparatively normal bowel function or even with constipation between times. During the exacerbations usually there are localizing signs in the appendix area such as tenderness and rigidity. Such attacks last only a week or two but keep repeating themselves at longer or shorter intervals until their cause is recognized and corrected. Even though there may be no diarrhoea during remissions, digestion frequently is impaired, physical examination and x-ray films will reveal abnormal signs about the cæcum and appendix, and so it is possible to explain why the colitis keeps recurring.

*Cancer of the intestine* may give rise to chronic diarrhoea early in its course or only after gradually increasing obstruction. This obstruction, leading to accumulation behind the site of narrowed lumen, causes ultimately a compensatory diarrhoea to rid the bowel of the retained fæces by copious and repeated discharges. Thus there develop alternating periods of constipation and diarrhoea resembling the course of chronic catarrhal colitis. Cancer of the rectum particularly may give rise to a chronic diarrhoea persisting for weeks or months before

other more definite symptoms and signs lead to recognition of the real cause. Neoplasm at any other site, such as sigmoid colon, one of the flexures or the cæcum, may similarly disturb bowel function with increased frequency of discharges like those observed in chronic catarrhal colitis, and this may go on for some time before such additional manifestations as pain, toxæmia, anæmia and cachexia call attention to the serious nature of the patient's illness. Sooner or later also the stools may change in character because the cancer has ulcerated and discharges blood into the bowel from its surface. This development with discovery on physical examination of a palpable tumor somewhere in the abdomen and demonstration by x-ray films of a break in the continuity of the bowel, taken in connection with the previous history, all assist in making the diagnosis finally clear.

*Chronic venous stasis* in the bowel wall as a cause of chronic diarrhœa is observed frequently when cirrhosis of the liver has led to portal obstruction, or may occur also as a result of impaired vis a tergo in chronic valvular or myocardial heart disease. Other symptoms and signs besides disturbance of bowel function should be recognized, if careful investigation is made.

Finally there is another form of chronic diarrhœa resembling chronic catarrhal colitis but due solely to *intestinal neurosis*. This occurs in high-strung, nervous, excitable and hysterical individuals, especially in women. They may have normal bowel function part of the time, but after any emotional strain or prolonged excitement or disagreeable experience diarrhœa appears and persists for days or weeks in spite of all kinds of treatment. That there is such a type of bowel disturbance has long been recognized. It is usually obstinate and intractable,

but it is not due to chronic inflammation of the bowel, is purely nervous in origin and never proves fatal.

*Treatment.* — In the management of chronic catarrhal colitis, diet is the matter for first consideration. Unless one habitually questions these patients about what they eat, how much they eat and at what hours, it is incredible how little regard they have for their digestive tract. Improper kinds of food, too large quantities at a meal, irregularity about the time of eating, are the cause of most of the disturbances of bowel function classified as chronic catarrhal colitis. The first duty of the physician, therefore, is to regulate what these patients eat, to instruct them as to what constitutes a proper amount of food and to impress upon them the necessity of regular hours for meals. Unless this is done, no other measures are of much use. The diet should be soft, bland and with slight residue. A list such as that recommended for chronic gastritis may be used as the basis also for chronic catarrhal colitis, though it may have to be modified and restricted as regards fruits, fruit juices and salads, if too frequent bowel movements persist. Food stuffs highly bacterial or toxic, such as meats or game kept too long, cheese rich in the chemical products of putrefaction and highly seasoned sauces of all kinds should be carefully eliminated. Simple, uncomplicated dishes, properly cooked, are essential to success in treating this disease. Even proper foods do harm, if eaten to excess, so that many courses and second helpings should be forbidden. Finally there must be regular hours for meals, so spaced as to allow opportunity for digestion and absorption of food, and no “piecing” between times or late suppers in addition to the three regular occasions commonly adopted for eating. Such rigid attention to rules must be continued until the irritable bowel has become

less active and the number and character of the discharges more normal. This may be a matter of weeks, and no rapid recovery should be expected.

Next in importance to diet is rest, both physical and mental. The patient should try to obtain twelve hours out of the twenty-four in bed, say from 8 or 9 P.M. to 8 or 9 A.M. strictly avoiding dissipation at night clubs, dances, receptions and cocktail parties, where food and alcoholic liquors as well as excitement and overexertion are factors in preventing a cure. During the twelve hours out of bed all excessive activity should be restricted, such as long walks, tennis, golf, swimming, dancing, bowling and horseback riding, and all intense expenditure of nervous energy incident to business or to social affairs should likewise be shunned.

There are no drugs that are specific in chronic catarrhal colitis, and those that are employed are mainly symptomatic. But any remedy that acts by limiting peristaltic activity in the bowel or by restricting the secretion of mucus is apt to do more harm than good. For this reason opium, acetate of lead, nitrate of silver and all the gallic and tannic acid preparations, formerly recommended, are no longer in favor. If gastric analysis shows achlorhydria, dilute hydrochloric acid in twenty drop doses after each meal sometimes will check diarrhœa, provided at the same time all the precautions about diet and rest are carefully observed. Remedies injected into the colon through the anus and not made to reach it through the stomach and small intestine are a more rational method of therapy. But even these usually are not of sufficient benefit, when used by the patient at home, to justify the trouble and discomfort they cause, however valuable they may be when administered in a hospital by trained assistants.

Most cases of chronic catarrhal colitis respond satisfactorily to dietetic treatment with rest, as described. If they do not, then further search should be made for other conditions that underlie the bowel inflammation and are factors in its production. If heart weakness is discovered, digitalis should be added to the therapy. If neurosis appears to play a part, bromides will aid in controlling the symptoms. The possibility that some more serious disease of the bowel, such as chronic appendicitis or neoplasm, is responsible for the chronic diarrhoea, must always be borne in mind, and careful search made for it. Finally, it is always possible that some one of the agents that produce ulcerative colitis may be active in less than usual intensity, even though mucus only and no blood is found in the stools at the time the patient is under observation.

### *Chronic Mucous Colitis*

This is an unusual form of intestinal disease, not often seen. The name colitis assigned it is unfortunate, because it is doubtful whether the condition is due to chronic inflammation at all, and the descriptive term "mucous" is more applicable to the other non-ulcerative form just considered than it is to this one. Consequently confusion constantly arises as to what is meant by chronic mucous colitis. Other names by which it is known are membranous colitis and myxo-neurosis intestinalis. The infrequency of this ailment needs to be emphasized. It is undoubtedly a rare disease as compared with chronic catarrhal colitis.

*Clinical History.* — The peculiar and characteristic feature of so-called chronic mucous colitis is the occurrence of attacks of severe colic from time to time, followed by the passage of mucus in strips or sheets, sometimes in tubular casts, as if the bowel

had shed its inner lining membrane. The pain is paroxysmal and intense, and the amount of mucus passed at the time is often surprisingly large. Such attacks are infrequent, may last for several days or a week, are accompanied by great soreness and sensitiveness of the abdomen but rarely by fever of any consequence. Usually they are induced by physical or mental exhaustion or some violent emotional disturbance. Between times there may be no diarrhœa at all. In fact constipation may prevail instead.

Certain other features in the clinical history besides the peculiar attacks of colic aid in identifying the disease. The patients usually are women much more often than men. They are all abnormal as regards their nervous systems, usually excitable, inclined to be hysterical or else depressed, gloomy and melancholy. There is also frequent association of the bowel attacks with other abnormal physical conditions, such as visceroptosis, asthma, urticaria, chronic menstrual difficulties in women with severe dysmenorrhœa and chronic sexual complaints in men. All these patients are introspective, self-centered, neurotic and hypersensitive, and most of them conscious of their bowel and complaining about its behavior.

*Physical examination* is quite negative in these cases as regards the abdomen except for tenderness over the lower part and along the course of the colon. Greater tenderness discovered over one area than another may lead to error, unless it is remembered that all tenderness is a subjective sign, and that these patients do not always exercise good judgment in deciding. Furthermore, while the bowel may be spastic at times so that it can be rolled under the examining fingers, no tumor mass is ever palpable, if the condition is only mucous colitis.

More is required than localized tenderness and a palpable bowel to recognize either chronic appendicitis or neoplasm.

*Stool examination* may show no mucus in excess of normal amount during the interval between attacks. But when an attack occurs of the peculiar violent paroxysmal colic, to which these patients are subject, the material discharged from the bowel and brought for inspection is sufficiently characteristic to make the diagnosis unmistakable. It consists of large strips and sheets and even tubular casts of the bowel that are not seen in any other condition. These are particularly impressive when floated on water and spread out.

*X-ray Examination.* — The colon usually is irritable and quickly expels the barium injected. It is contracted, smooth and spastic. But while the x-ray films thus prove that colitis is present, they do not determine its character. They do eliminate, however, such possibilities as chronic appendicitis and neoplasm.

*Proctoscopic examination* reveals no ulcers and no characteristic abnormality that helps to identify the kind of colitis from which the patient suffers.

*Diagnosis.* — To sum up the evidence, violent attacks of abdominal pain followed by the discharge of large amounts of mucus from the bowel in visible strips, sheets and casts is the characteristic feature of membranous or mucous colitis. The attacks occur usually in a woman with configuration of body, poor nutrition and neurasthenic traits so common in visceroptosis, or else in a woman, who without deviation from the normal physical development, is high-strung, neurotic and excitable. Such a combination of incidents is practically diagnostic. In reaching a conclusion, however, it should be kept in mind that this disease is unusual and not often en-

countered. Every care about investigation, discrimination and judgment should be exercised before this conclusion is accepted.

*Treatment.* — The one great danger is that the sudden violent attack of abdominal pain will be misinterpreted and lead to needless laparotomy for some condition suspected but not present. Sufficient delay to permit accurate observation and investigation will prevent this error. In the management of these cases attention should be directed to the general constitutional incapacity rather than to the bowel. Rest in bed for weeks, a bland diet without residue, attempts to build up body weight, sedatives such as bromides as may be required for nerve storms, all methods of hydrotherapy, physical therapy and psychotherapy, found useful in neuræsthenia, are the remedies of greatest value. It must be admitted, however, that these cases are most difficult to manage and sometimes defy all plans suggested for cure.

# CHAPTER XV

## CHRONIC ULCERATIVE COLITIS: AMŒBIC

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This differs from the types of colitis so far considered by the presence of ulcers in the bowel, as the name implies, and by their tendency to bleed and so to produce blood in the stools. Several different groups are recognized, all characterized by ulceration but not all due to the same cause. It is possible from the standpoint of etiology to differentiate four different subdivisions, known as amœbic, bacillary, tuberculous and a fourth group due to some unknown cause, not like any of the others in clinical history or pathology, classified simply as non-specific ulcerative colitis. Blood in the stools is a distinguishing sign between all these groups and chronic catarrhal colitis. But blood may not be constantly visible on gross inspection, even when occult blood can be demonstrated by chemical tests. In exacerbations, however, such as occur in all varieties of chronic ulcerative colitis, blood is passed with the bowel discharges in greater or less amount for a variable time and constitutes one of the characteristic features.

Chronic amoebic colitis is the variety of chronic ulcerative colitis most often seen on the Pacific Coast. Infestation by the *Entamoeba histolytica* begins in the large bowel but may extend from there to the liver. In the intestine it causes recurring attacks of acute diarrhoea or dysentery with the passage of numerous discharges containing blood and mucus, but there are intervals between such attacks of lengthy or brief duration, when there may be no diarrhoea at all, and on the contrary constipation may occur. It is a most serious and obstinate disease, not only causing intense suffering at times but also in some instances a fatal outcome.

The amœbæ or their cysts are taken into the body with food or drink, most often with uncooked vegetables, contaminated by human fæces used as a fertilizer, or with impure water from wells situated too near privy vaults and thus contaminated by underground seepage, or possibly with any food that has been contaminated by the hands of those who prepare it in hotel or restaurant or home, because they are carriers. How long amœbæ remain in the body before symptoms develop has never been accurately determined. The period of incubation apparently is a variable one. It may be one week, or it may be as long as six. Usually there is no consciousness of any ill-health until the sudden onset of an acute attack, but vague symptoms of general malaise may precede or even a mild diarrhoea.

### CLINICAL HISTORY

When this sudden attack makes its appearance, it is characterized by severe abdominal pain, frequent loose bowel movements becoming semi-fluid or fluid, more or less constant desire for an evacuation, or tenesmus as this symptom is called,

and in extreme cases the passage of blood-stained mucus or a bloody fluid in small amounts with no relief. After a few days with discharges varying in number from six to eight up to fifteen to twenty each day the patient becomes exhausted by pain and loss of sleep. There is no fever as a rule, but slight elevation of temperature to about  $100^{\circ}$  F. may be observed. Usually the temperature is normal. Such attacks last four to five days or a week and subside gradually from their original intensity; but others follow similar in character after an intermission of weeks or months. During these intervals the bowels may continue abnormally loose and the discharges frequent, and the stools may persistently contain mucus and blood in variable amounts, but there are none of the violent symptoms of an acute attack. On the other hand during the intermissions abdominal discomfort and uneasiness and vague disturbances of digestion may constitute the only reminder of illness, and the stools may be formed and apparently normal in character on gross inspection. In some instances constipation replaces diarrhoea between attacks, but more often there is complaint of sudden desire and necessity for haste in evacuation that show the bowel is irritable and unduly sensitive. During these periods of comparative freedom from the usual symptoms of chronic colitis patients are nevertheless in a condition of amœbic infestation, continue to pass the organisms and cysts in their stools and are thus a constant menace to those about them and a potential source of infection.

The most important complication of the disease is transference of the amoebæ from the colon to the liver by the blood and the formation there of a tumefaction that ultimately becomes an abscess. This new development is made known to the patient by pain and tenderness in the liver region and

possibly by the discovery of a palpable lump below the ribs. A story of previous bowel disturbance may or may not be obtained. Usually the liver complication follows a chronic diarrhoea but not necessarily a violent dysenteric attack, but no history of either may be elicited. It is important to keep this fact in mind, that the bowel condition may remain entirely latent until the first symptoms to attract attention are those in the liver region, or that months or years may elapse following the original acute attack of amoebic dysentery before the symptoms of liver abscess arise. When they do appear, there may be no severe pain at the outset, but only an annoying discomfort which gradually increases.

### PHYSICAL EXAMINATION

*Physical examination* reveals little of value in diagnosis. As in every form of colitis the abdomen is more or less tender along the course of the large bowel and in this form particularly during or just after an exacerbation. Usually the tenderness is greatest over that part of the colon most affected, and this is most frequently the cæcum and ascending colon. Muscle spasm over this area as well as maximum tenderness there helps to identify the site of the most active ulcerative lesions. A rounded swelling may be seen bulging out the right costal margin when abscess of the liver is a complication. But such enlargement is not always demonstrable even when amoebæ have invaded the liver.

### PROCTOSCOPIC EXAMINATION

*Proctoscopic examination* supplies information, not obtainable in any other way, about the presence of ulcers and their appearance. If ulcers are visible and are caused by

amœbæ they are, as a rule, discrete, more or less oval, have ragged edges, their margins are undetermined, and their floor is covered with a yellowish slough. They may be present in large numbers in the sigmoid colon within reach of inspection through the instrument, but may not appear so low in the bowel, though present in the cæcum and upper colon too high for observation. Material obtained directly from an ulcer base through the sigmoidoscope usually will show amœbæ even though they can not be found in the stools. While dysentery is active or soon after an acute attack, instrumentation of bowel is too painful to justify its employment, but in the interval between attacks its use is permissible. However, the procedure is always a disagreeable one, and in private practice it is well to postpone it until after careful stool examination. If by this method of investigation amœbæ or cysts are found, proctoscopy is not needed for diagnosis.

#### LABORATORY EXAMINATIONS

Certainty in diagnosis depends upon demonstration in the *stools* or in material obtained through the proctoscope from the base of an ulcer in the large bowel of the *Entamæba histolytica*. But these organisms are not always easy to identify. There are at least five different varieties of amœbæ that live in the human intestine and only one is the cause of this disease. Upon its recognition and identification diagnosis rests. Examination of a fresh specimen of fæces may be made under the microscope directly when the organism is motile, or after staining with Lugol's solution, as described in the chapter on methods of investigation in intestinal disease. The true *Entamæba histolytica* cyst seen in the stained specimen has only a few nuclei, two to eight, with four as an average number,

while the other varieties have more, from eight to sixteen. Even so it is easy to err, and the practicing physician, unless he has had special instruction about how to differentiate one variety from the others, should obtain the opinion of an expert laboratory technician before beginning treatment for amœbic colitis. Many stools may have to be examined before certainty is reached. One specimen proves nothing, either for or against.

The *blood* shows anæmia, as a rule, in chronic amœbic colitis with the red cells between three and four millions, though they may be even lower. The hemoglobin is reduced in proportion. The white cells are increased in most cases but not excessively, with an average twelve to fifteen thousand. But there is no count and no picture in the blood smears that is characteristic or diagnostic.

#### X-RAY EXAMINATION

*X-ray examination* after a barium enema shows the usual abnormalities in the colon that prove the patient has colitis but none that identify this as the amœbic form. In two ways, however, x-ray films are of value. First, when the symptoms and clinical history point to chronic colitis, but no amœbæ are found in the stools, the films at least eliminate the possibility that a neoplasm of the bowel is responsible. Second, when amœbæ are found, but specific treatment does not promptly give relief, x-ray films of the colon may demonstrate that some other pathological change is present as well as the changes produced by amœbæ.

#### DIAGNOSIS

Typical cases are easily recognized. The history of recurring attacks of acute dysentery and the finding of the amœbæ in

the stools make the diagnosis positive. If doubt still exists, proctoscopic examination with demonstration of characteristic ulcers and amoebæ in the smears made directly from them affords final proof. But the atypical or latent cases are the ones that cause trouble. In these there may be no attacks of dysentery in the present or past history; the stools may show no amoebæ or cysts in spite of repeated search, and the proctoscope may fail to reveal ulcers because they are situated in the bowel above the point the instrument reaches. In such cases Epsom salts by mouth may, by causing fluid evacuations, remove some of the parasites from the bowel wall, and numerous examinations for them must be made at different times before the search is abandoned. Any chronic diarrhoea, especially if the stools contain blood, and any chronic disturbance of digestion, however vague, may be due to chronic amoebic colitis, and that possibility must be kept in mind.

Usually there is no difficulty about recognizing an ulcerative colitis during an exacerbation, for the pain, the tenesmus, the numerous bowel movements and the occasional or constant presence of blood as well as of mucus in the discharges make the condition unmistakable. But even at the time of an exacerbation the problem remains as to cause. Two other varieties resemble amoebic colitis in the frequency and the character of the dysenteric attacks they produce, the bacillary and the non-specific. To identify these and separate one from the other requires microscopical and cultural examinations of the stool. The fourth variety of ulcerative colitis, the tuberculous, does not as a rule give rise to such acute, severe, painful attacks of diarrhoea as are seen in the other three, because the ulcers do not form so far down in the bowel as in the other varieties and are apt to be limited to the ileocæcal region.

When the history does not include the violent disturbances of intestinal function known as dysentery, but the complaint is only of recurring attacks of diarrhoea from time to time, more or less constantly as in chronic catarrhal colitis, with abdominal discomfort and gas associated with a lack of energy and possibly joint and muscle pains, the diagnosis of chronic amoebiasis may be overlooked, unless the lower bowel is inspected by the proctoscope, and the stools are examined carefully and repeatedly for amoebæ and cysts. The stools may appear normal or even constipated on gross inspection over long periods of time, and yet these patients may be carriers of amoebæ and a menace to others with whom they associate. The bowel condition thus is not always a dependable guide. In all obscure digestive ailments or vague disorders of general health it is therefore advisable to have a routine stool examination made, including the search for amoebæ and cysts, before final diagnosis is reached.

### TREATMENT

So many different remedies have been suggested for ridding the intestine of the *Entamæba histolytica* and its cysts that it is difficult for the general practitioner to decide which to select. Personal experience with the following plan has convinced the writer of its efficacy, at least at the outset of treatment, because it produces results quickly. For six mornings in succession an ampoule containing one grain (60 mgm.) of emetine hydrochloride is given hypodermatically. These can be obtained ready for administration. At the same time for four nights in succession the patient is given by mouth one-quarter grain (15 mgm.) of emetine bismuth iodide, then for four nights one-half grain (30 mgm.) and then for four nights one grain

(60 mgm.). This drug usually is dispensed in tablets but gives better results if each tablet is crushed to powder and enclosed in a capsule. The whole course of treatment thus occupies twelve days, the first six with emetine injections and small doses by mouth of emetine bismuth iodide; the second six with larger doses of emetine bismuth iodide by mouth but no injections.

There is no doubt that at the time of an exacerbation of the disease with pain and dysentery no drug acts so promptly as emetine in giving relief. Furthermore it has no bad effects in the doses prescribed. It may cause nausea, but usually this is slight. Unfortunately it can not be relied upon to destroy all the parasites in the bowel by one course such as described, and unless the course is repeated, or some other remedy is given to supplement it, sooner or later relapse is likely to occur. For this reason it is better, after sufficient time has elapsed to allow the patient to recover from any possible disagreeable effects of emetine, to repeat the course as described. Usually the interval between these two courses need not exceed one month. If it is decided to substitute some other remedy to supplement emetine, in the opinion of the writer carbarsone holds first place. This is supplied in capsules, each containing three and three-quarters grains (.25 gram). One such capsule twice a day, morning and evening, for ten days, or five grams altogether, constitutes a course. This preparation contains arsenic and should not be repeated too promptly. A ten day interval at least should be allowed between courses. Carbarsone appears to be more efficacious than any other remedy in permanently ridding the intestine of amœbæ. But other drugs are still preferred by the best authorities. Craig recommends chiniofon, a preparation containing

iodine as its active agent. A tablet of three and three-quarters grains (.25 gram) is given three times a day for fourteen days. The Mayo Clinic recommends treparsol, another member of the arsenic group, likewise in tablet of .25 gram, three times a day for four days, repeating the course if necessary at ten day intervals. Amoebiasis has an unfortunate habit of relapsing weeks or months after it has apparently been overcome, no matter what plan of treatment has been employed. It is advisable, therefore, to examine the stools from time to time after clinical cure and to repeat treatment if amoebæ or cysts are found.

The following history illustrates the clinical course, response to treatment and tendency to relapse that characterizes an average case of amoebic colitis.

A Japanese farm laborer, aged 36, came to San Francisco for medical advice in 1923, because for three years he had trouble with his bowels consisting of several movements each day containing blood and "slime", passed with pain and straining. For two weeks preceding his visit he had noted also pain in his right side at the edge of the ribs with a sense of swelling there. He appeared ill and weak. No abnormality was found in heart or lungs. In the right upper abdominal quadrant, just below the liver and apparently a part of it, there was a visible and palpable tumor, the size of a small orange, very tender, rounded and smooth. The abdomen otherwise was normal except for tenderness in the course of the colon. There was no fever and no increase in pulse rate. The stool consisted entirely of bloody mucus, showed many large, active amoebæ containing red blood corpuscles, and also numerous cysts of *Entamoeba histolytica*. The blood count was that of a moderate secondary anæmia with leucocytes 11,700. On routine treatment by emetine injections and emetine-bismuth-iodide by mouth as described not only his amoebæ and cysts disappeared and his stools became normal, but the liver tumor rapidly decreased in size, until

it was no longer palpable. This patient remained well until 1926 when a recurrence of his diarrhoea with the passage again of bloody mucus brought him back for further advice. Again amoebæ and cysts were found in his stools, again the same treatment was given, the symptoms disappeared, and the stool became negative. This time he remained well until 1934, when he reported, that for three months his trouble had recurred. At this third visit amoebæ and cysts were found again, the treatment was repeated, and a clinical cure was effected promptly. He has not been seen since, though urged to report for stool examination before another relapse developed.

The following case is an example of the latent type of amœbic colitis, not to be identified by symptoms alone.

A man, aged 53, seen first in 1922, complained of distress in his upper abdomen running up under the breast bone and to the left side of his neck. This was not constant, but came in attacks at long intervals for two years past. All attacks appeared to occur in consequence of certain foods eaten, particularly green vegetables. They came on two or three hours after a meal, lasted for several hours, once for two days. The description he gave of them was vague, but there was no pain, no nausea, no vomiting, and relief was obtained by baking soda and the belching of gas. For four months before his visit he had noted a sense of swelling or bulging just below the ribs on the left side at intervals, most often at night, lasting an hour or two, then passing away spontaneously. The bowels moved regularly once each day, and he never had diarrhoea. No abnormality was found on physical examination. Gastric analysis showed only a hyposecretion a little below the normal average. The blood count was normal and Wassermann negative. The urine showed no abnormality. Treatment by diet, exercise and stimulants to gastric secretion did not relieve the patient's symptoms. Finally after three months, in spite of a history of normal bowel function, the stools were examined. Cysts of *Entamoeba histolytica* were then found. Routine emetine treatment was given during July and August 1922, and the symptoms all disap-

peared. The patient remained well until January 1923, when he returned complaining of the same sense of abdominal bloating with pressure extending up under the breast bone and other symptoms as described at the outset. Stool examination again showed many amœbic cysts. After another course of emetine treatment the symptoms all disappeared, and no more amœbæ or cysts were found in his stools. There has never been any return of his abdominal trouble from that time to this, and repeated stool examinations since, at least once every year, have failed to show any amœbæ or cysts.

## CHAPTER XVI

### CHRONIC ULCERATIVE COLITIS: BACILLARY

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#### CLINICAL HISTORY

This form of intestinal disease is not so common in temperate climates as that due to amœbæ, but the two resemble each other in making their first appearance usually by an acute attack in a patient previously well. The onset is sudden and as a rule violent with severe pain in the abdomen, tenesmus and frequent bowel discharges containing bloody mucus. So far the two diseases are alike. But in the bacillary form there is more fever, a rapid pulse and greater evidence of prostration. This acute illness lasts for a week or ten days in most cases but possibly longer, and while it may then subside completely and leave the patient well, many times it persists in a less violent form as a chronic disturbance characterized by abnormally frequent bowel movements, unformed stools, uneasiness and discomfort or even actual pain in the abdomen. From time to time also after errors in diet or physical overstrain or for some reason unknown relapses occur like the original attack. Thus

there are three possible outcomes of the first original illness; complete recovery after a stormy course of a week to a month, or a fatal termination within a few days, or gradual improvement without complete restoration to normal and a chronic condition persisting for months or years with intercurrent exacerbations.

The disease is widespread in its incidence. This is proved by the fact that our knowledge regarding the micro-organism that causes it has been furnished by the researches of Shiga of Japan, Flexner of the United States but working in Manila on this problem and Kruse of Germany. This micro-organism commonly known as the Shiga bacillus, because he first described it and identified it as the specific agent in this form of dysentery, enters the body with food and drink and leaves it in the bowel discharges. Food contaminated by the hands of chronic carriers of the infection is a common source of the disease.

The period of incubation is short, as brief in some instances as one or two days, usually less than a week. While in the acute attack the symptoms of bacillary dysentery resemble closely those of amœbiasis as already stated, the toxæmia is greater, as shown by higher fever, more extreme prostration and in general a more acute course. But the clinical features alone do not differ sufficiently to distinguish this disease from the amœbic variety, and if dependence is placed upon history, the only diagnosis possible in the acute attack is ulcerative colitis without specifying the type.

In the chronic cases likewise there is nothing in the clinical course to separate one from the other except that the story is apt to be one of more constant impairment of bowel function between acute attacks in the bacillary without the periods of

comparative freedom from annoyance that are allowed by the amoebæ.

### PHYSICAL EXAMINATION

By *physical examination* all that is found is a tender abdomen, more or less sensitive as the condition is active or quiescent, but with no characteristic sign to distinguish this from other varieties of ulcerative colitis. The temperature is always moderately elevated, but never extremely high. It may reach 103° F. but is not always above 100° F. The pulse usually is rapid, its rate varying with the degree of toxæmia and the severity and duration of acute symptoms.

### PROCTOSCOPIC EXAMINATION

*Proctoscopic examination* is never justified during an acute attack because of the extreme tenderness and irritability of the anus and rectum. But in the chronic stage, between acute exacerbations, it may be used as an aid to diagnosis, if this remains uncertain in spite of all other methods of investigation. Ulcers, if present at all, usually are low enough in the colon to be seen and identified. But they may be situated mainly above the sigmoid and therefore inaccessible for inspection, or in the chronic case there may be no ulcers because they have been replaced in healing by granulation tissue. They are described as bleeding easily, irregular in outline with no undermining of the edges like amoebic ulcers, numerous but superficial, tending to become confluent and with generalized inflammation of the intact mucous membrane between them, which presents a granular appearance. There appears on the whole to be considerable doubt among authorities about the value of proctoscopy in identifying the ulcers of this form of colitis.

## LABORATORY EXAMINATIONS

Only by the laboratory can a positive diagnosis of bacillary dysentery be made. The stools during an acute attack are thin on gross inspection, light in color, muco-purulent in character, occasionally blood streaked, differing from those in amoebic dysentery, which usually are thicker, consisting of masses of mucus and dark decomposed blood. Microscopically they show many red blood cells, polymorphonuclear leucocytes and desquamated epithelial cells but no *Entamæba histolytica* and no other organisms that can be identified by direct inspection under the microscope. The causative bacillus must be recognized by its characteristics in cultures and its response to fermentation tests. But these cultural characteristics are variable, because there are different strains of the organism, and all do not give just the same response. The problem of identification thus is never an easy one even in the research laboratory. Bacilli are present in larger number during the acute attack and more easily recognized than during the quiescent chronic stage, but at any time bacteriological examination affords the only proof of their presence.

The bacilli of this disease do not enter the blood stream from the bowel wall as those of typhoid fever do, so that blood cultures give no aid in diagnosis. But anti-bodies are formed, as a rule, though not always, and usually not before the tenth day of the disease at best. Agglutination tests thus become of value but present many practical difficulties. The various strains of the Shiga, Flexner and Kruse bacillus will give the reaction but only when the patient's blood serum is added to the right stock culture, and many may have to be tried before the right one is found. The reaction therefore may never be

obtained at all, simply because the right strain has never been used. Furthermore it seems to be agreed that unless the agglutination persists in high dilutions it has no significance. Positive reactions in dilutions up to 1 to 100 either do not occur at all, or if they do, can not be accepted as indicating specific infection. Finally, the various strains of the bacillus respond to differing dilutions so that there is never any certainty as to what the reaction means, even when it is found. All these peculiarities make the agglutination test unsatisfactory and not altogether trustworthy in bacillary dysentery.

### DIAGNOSIS

In any case of chronic ulcerative colitis, recognized as such by its clinical history, the Shiga bacillus or one of its strains may be the cause. The fact that the trouble began with a violent acute onset, that subsequent intestinal disturbances followed as a sequel, and that there have been frequent or occasional more or less violent exacerbations throughout weeks or months since the original attack, always makes it probable that the cause is either the *Entamæba histolytica* or some strain of the Shiga or Flexner bacillus. As the acute attack is commonly the first occasion on which the physician meets the patient, it is upon the clinical features of this attack that dependence must largely be placed to distinguish one variety from the other. In acute bacillary dysentery, the generalized effects are more sharp and severe with higher fever and greater toxæmia. The local changes in the bowel produced by the bacilli result in bowel discharges that are more liquid and profuse, while those produced by amœbæ result in smaller, semi-solid discharges of mucus and blood. Microscopical

examination of the stools in bacillary dysentery shows red and white blood corpuscles and desquamated epithelium but no amœbæ, while in the amœbic variety the parasites should be found during the acute attack. Cultures from the stool should demonstrate bacilli with the characteristics as regards morphology and effects upon media that belong to the Shiga-Flexner organism. Sometimes this is easy but more often difficult because of the many variations shown by different strains. Finally agglutination tests should give positive reactions in high dilutions. But here again results are often disappointing and not always dependable either for or against diagnosis. If the acute attack resembles clinically amœbic rather than bacillary infection, but the diagnosis is unsettled, an ampoule containing one grain (60 mgm.) of emetine hydrochloride may be injected each day for several doses as a therapeutic test. A chronic case, that first comes under observation weeks or months after the original acute attack, offers much greater obstacles to differential diagnosis. Dependence must then be placed more upon the laboratory than upon clinical observation, and the results of stool examinations and of agglutination tests are the main hope. It helps a little, however, to realize that in chronic bacillary dysentery the symptoms are likely to be more continuous and in chronic amœbic more intermittent. But this rule also is not always reliable.

### TREATMENT

(a) In the acute stage the symptoms ordinarily are so violent, the suffering so severe and the prostration so rapid and pronounced, that it is not advisable to wait for complicated laboratory examinations by stool culture and blood agglutination tests to establish the exact nature of the intestinal

disease before treatment is commenced. This should be conducted at the outset on the same plan as that advised for acute inflammation of the intestines, regardless of etiology. The patient should be placed at rest in bed. As soon as possible, one ounce (30 c.c.) of castor oil should be given. If the patient is vomiting and can not retain the oil, then calomel and soda powders should be substituted, one-quarter grain (15 mgm.) of calomel and one grain (60 mgm.) of soda every half hour until at least six such powders and possibly twelve have been given. If the stools after castor oil or calomel still show fecal material, magnesium sulphate in saturated solution should be administered, one teaspoonful every hour until no further food refuse appears in the bowel discharges. The diet meantime should be exclusively liquid, mainly weak broths and cereal gruels, avoiding milk as previously suggested in the treatment of non-specific acute inflammation. After the oil or the calomel or the magnesium sulphate have rid the intestine of contents present at the outset of the attack, remedies should be directed to checking peristaltic activity and relieving pain. For this purpose the same opium and bismuth mixture recommended before should have first choice, regulating the repetition of the dose by its effects on bowel evacuations. Camphorated tincture of opium may be substituted in the mixture for the plain tincture if there is much prostration, in dose of one drachm (4 c.c.) of the former instead of five minims (0.3 c.c.) of the latter. Incessant desire for bowel evacuation is best overcome by a rectal suppository of one grain (60 mgm.) aqueous extract of opium and one-quarter grain (15 mgm.) extract of belladonna in cocoa butter, or ten to twenty minims (0.6 to 1.3 c.c.) of the tincture in one or two ounces (30 to 60 c.c.) of starch water. Heat to the abdomen is another valuable aid in

relieving pain, applied by hot water bag, hot fomentations or electric pad.

While this treatment is being carried out for ulcerative colitis regardless of what produced it, laboratory investigation should likewise be in progress to discover the cause. If this proves to be the Shiga bacillus or one of its strains, then the usefulness of a specific serum must be considered. A polyvalent preparation to include several strains of the different types is supplied under the name anti-dysenteric serum. The dose advised runs as high as 60 to 100 c.c. intravenously once daily for two or three days. However, its effects on the disease are uncertain, and no assurance can be given that they will be favorable in the case to be treated, even though they have proved so in others. On the other hand serious shock may follow and serum sickness, as in the use of any of the biological agents, requiring the administration afterwards of adrenalin chloride solution, 1 to 1000, in dose of one-half to one c.c. subcutaneously. Altogether the value of this procedure must be weighed carefully in connection with the seriousness of the patient's condition. If this seems desperate, taking chances is justifiable; if not, simpler measures of treatment may suffice.

(b) In the chronic stage, when the disease has been allowed to run on for months with recurring exacerbations but never at any time complete recovery, the bowel movements remaining soft, muco-purulent and too frequent even during the intermissions, and the general condition of the patient becoming gradually one of malnutrition and secondary anæmia, the first requirement is to determine, if possible, whether the Shiga bacillus or some of its strains is still responsible. The effort then to eradicate this infection must be accompanied by other therapeutic measures to improve the patient's resistance and

build up the blood. It is not always possible in these chronic cases by any method to rid the patient of his persistent disturbance of bowel functions. Specific bacilli may become few or absent from the stools, but the symptoms persist. It has been suggested that some of the cases, ultimately assigned to the group known as non-specific ulcerative colitis, where no causative organism can be identified, have begun as bacillary dysentery and then have gradually changed in character because of secondary invasion of the intestinal mucous membrane by other forms, particularly streptococci, displacing the Shiga-Flexner group. At any rate, some of the cases recognized as bacillary in the original acute attack have finally reached a chronic state where no remedies did any good except those discussed later on in the consideration of non-specific ulcerative colitis, including surgical interference to permit prolonged irrigation of the bowel through an ileostomy or appendicostomy or colostomy.

A woman, aged 62, was brought to the hospital for treatment because for two weeks she had had a persistent diarrhoea with severe cramps low in the abdomen followed by liquid stools containing mucus and blood. Such discharges occurred about five times every day. She had no appetite, had grown weak and pale and had lost considerable weight. Previous to this attack she had been in good health. On physical examination the patient was found to be a small, emaciated woman with dry tongue and skin and low blood pressure but with no evidence of organic disease except diffuse tenderness over her abdomen. Her temperature on admission was 100° F. and ran thereafter an irregular course, at times normal, at times as high as 103° F. The stools were liquid, contained mucus and blood, but no amœbæ were found. Cultures showed a few colonies of non-lactose fermenting bacilli which gave sugar reactions of the Flexner organism. Tests with various members of the Flexner

group showed agglutination in dilution of 1 to 160 with Flexner Y and Flexner Z, and partial agglutination with Flexner Z in dilution 1 to 320. The blood count was low, the hemoglobin 58 per cent., the red corpuscles 2,750,000 and the white 2,950. The course of the disease was a stormy one. Forced liquid feeding, part of the time by gavage, glucose intravenously and Karo corn syrup by mouth, tincture of opium and tincture of belladonna more or less constantly to check peristalsis and relieve pain, symptomatic treatment for insomnia by amytal, all these led gradually to recovery after a month's illness. No serum was administered throughout. The patient's weight was gradually regained, and her blood returned to normal after her diarrhoea was checked, and she has remained well since.

## CHAPTER XVII

### CHRONIC ULCERATIVE COLITIS: TUBERCULOUS

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Ulcers in the intestine caused by the tubercle bacillus are found in the lower ileum, the cæcum, the sigmoid and the rectum but particularly and with greatest frequency in the ileocæcal region. It is not correct, therefore, to speak of tuberculous colitis with the understanding that the disease is widespread in the colon or that it is the only part of the bowel ever involved.

While it is true that the infection rarely is primary in the bowel and that in over 85 per cent. of the cases of tuberculosis of the colon there are active or healed lesions in the lungs, it must also be kept in mind that pulmonary disease does not invariably precede. Tuberculosis of the large bowel may occur without any symptoms referable to the lungs at the time and without any evidence of active or arrested disease there shown by x-ray films. In the investigation of 52 cases of tuberculosis of the cæcum observed at the Massachusetts General Hospital between 1924 and 1933, it was found that one-third of the

patients, who had this disease, presented no evidence either clinically or by x-ray examination of tuberculosis in the chest (New Eng. Jour. Med., August 1, 1935, p. 235). It can not be doubted, therefore, that primary intestinal tuberculosis does sometimes occur. When it is a complication of pulmonary tuberculosis, it is seen as a rule in fairly well advanced and not in incipient cases, so that the symptoms of pulmonary usually precede those of intestinal disease. But this is not invariably true, even though pulmonary tuberculosis really appeared first, for the disease of the lung may have given little or no evidence of its presence, and disturbances of bowel function may be the patient's first and chief complaint.

### CLINICAL HISTORY

The common symptoms are those noted in any case of ulcerative colitis, pain and diarrhoea. But no symptoms may appear until late even when x-ray shows the colon involved. There is no sudden and violent onset as in other forms of the disease. At first the pain may be slight and occur irregularly, but as time goes on it increases in severity and in persistency. As the most frequent site of pathology is always the ileocæcal region, pain is felt more often in the right lower quadrant than elsewhere. Bowel movements likewise vary in number, and there may be periods at first when constipation alternates with diarrhoea, during which there are no other symptoms. But gradually diarrhoea becomes constant. The stools, always abnormal in character as well as in number, contain mucus in excess at all times as well as blood occasionally. Blood in the stools, however, is not as prominent a feature in this as in other forms of chronic ulcerative colitis, and the amount is never large. The fact that the patient has never observed it

does not disprove that ulceration exists or that the cause is tuberculosis. On the other hand the statement that blood is seen frequently in the stools simply is proof that the patient has an ulcerative colitis but does not identify the variety. This condition of the intestines, varying in severity but never presenting the stormy exacerbations seen in amoebic and bacillary dysentery and never disappearing entirely, leads gradually to loss of weight and strength and failure of general health. It may go on for a long time before it leads to a fatal outcome, and its duration depends largely upon whether there is an active pulmonary lesion as well.

### PHYSICAL EXAMINATION

The abdomen frequently is abnormal in consistence with the feeling designated "doughy". It is sensitive to palpation along the course of the colon, most definitely of all over the cæcum, and tenderness may be elicited there when it is not found anywhere else. Over the right lower quadrant not only this tenderness but also muscle spasm, rigidity, even a vague thickening or palpable mass may be noted. But all these signs may be observed in amoebic ulcerative colitis, in chronic appendicitis or in cancer of the cæcum, so that by themselves they fail to identify the disease that produces them as tuberculous colitis.

It is possible, however, for this disease to present so few signs on physical examination of the abdomen that no assistance in diagnosis is obtained from this method of investigation. But whenever it is suspected that a chronic ulcerative colitis may be due to tuberculosis, careful physical and x-ray examination of the lungs becomes as important as that of the abdomen and may solve the problem.

## STOOLS

There is no characteristic appearance of the stools on gross inspection that tells the colitis is due to tuberculosis. They show mucus constantly and blood at times; occult blood may be found when none is visible. Smears properly stained may show no tubercle bacilli even when ulcers are present in the bowel, because none are thrown off into the contents from the wall. Even when they are found in the stools their significance is dubious, if the sputum contains them also, for they may have been swallowed. Microscopical examination of stained smears of the sputum for acid fast bacilli thus becomes of even more value than that of smears of the stool in determining whether a chronic ulcerative colitis is tuberculous.

## PROCTOSCOPY

The proctoscope may discover ulcers if they are low enough in the bowel, but frequently they are not. If seen, they are irregular in contour, usually multiple, large in size, with indurated and reddened edges and gray base. They do not bleed as easily as those seen in other varieties.

## X-RAY EXAMINATION

The most characteristic finding by this method of investigation is hasty peristalsis or hypermotility. The bowel is irritable as in all forms of chronic ulcerative colitis. The only distinguishing feature that suggests tuberculous instead of some other variety is observed when the ileocæcal region is the site mainly involved. Then the cæcum will not retain the barium long enough to give its outlines in the film. This is so often the case that, if the filled cæcum can be demonstrated, it

is probably not tuberculous. These peculiarities are quite the opposite of what is seen in cancer of the cæcum or chronic appendicitis, both of which cause delay in the emptying time of this part of the bowel. The demonstration by x-ray films of ulcers in the bowel is quite impossible, regardless of the cause, and abnormalities such as spasticity, absence of haustra and irregular filling may be seen in any variety of chronic ulcerative colitis and not only in tuberculous. In cases where narrowing or definite stricture of the intestinal lumen is shown by x-ray, the cause can be inferred to be tuberculous ulceration only when all other facts on history, physical examination and laboratory findings are also taken into consideration.

### DIAGNOSIS

The symptoms and signs of chronic ulcerative colitis can be attributed without hesitation to tuberculosis only when they complicate obvious tuberculosis of some other part, particularly the lungs. Unless some special locality in the intestine, such as the ileocæcal region, is involved by the patient's story and by the physical examination, and unless this is confirmed by x-ray films, there is no other definite evidence to justify the diagnosis. The proctoscope frequently does not reach high enough to demonstrate the presence of ulcers. No tubercle bacilli may be found in smears from the stools. Then further proof must be sought outside the abdomen by physical examination and x-ray films of the lungs and by microscopical examination of stained smears of the sputum.

Whenever the symptoms and signs point to disease in the right lower abdominal quadrant, other problems arise for solution. First, instead of tuberculosis, may the cause be *chronic appendicitis*? Because the patient complains of pain

over this area and of tenderness there on palpation, the first thought usually is of chronic inflammation. Even the fact that there is frequent disturbance of bowel function with recurring attacks of diarrhœa alternating possibly with constipation may be the result of chronic appendicitis. But there is then no history of symptoms involving the lungs, preceding or accompanying those that involve the bowel, no bloody stools are observed, and there has been no progressive loss of weight, color and strength since the intestinal disturbance began. Physical examination may show in either case in addition to tenderness over the cæcum a more or less palpable mass in that region, but not of a character that identifies its etiology. Signs of disease in the lung, however, and the finding of tubercle bacilli in the sputum go far toward supplying proof that the intestinal condition is tuberculous. Valuable aid in differentiation is supplied in most cases by x-ray examination. In tuberculosis involving the ileocæcal region, it is difficult to make the cæcum visualize because its irritability prevents its retaining the barium, while in chronic appendicitis, the cæcum and appendix not only fill but tend to remain full longer than they normally should. On the other hand, the lungs may show definite signs of tuberculous disease in the x-ray films, when only indefinite signs have been found on physical examination, but no such abnormalities occur in chronic appendicitis. If doubt still remains after such resort to usual diagnostic means, it is better to assume that the condition is tuberculous and avoid operation, at least, while awaiting developments and making further study of the patient.

Second, may the symptoms and signs in the right lower quadrant mean *cancer of the cæcum*? This question can not be answered by clinical history only but can be usually by con-

sidering this in connection with physical examination and x-ray films. The tumor produced by malignant disease is hard, fixed and irregular as compared with that noted in tuberculosis of the bowel or chronic appendicitis. Furthermore, after a barium enema the cæcum visualizes readily and shows extensive filling defects not seen in tuberculous ileocolitis. There are also no evidences of pulmonary disease found by physical examination or x-ray films, when cancer of the cæcum is the diagnosis unless metastasis to the lungs has taken place.

### TREATMENT

Chronic tuberculous colitis is a most serious disease and every known resource sometimes fails to arrest its course. The means available for its control are the following:

(1) *Diet.* — With the wall of the colon containing ulcers throughout a considerable part of its extent and with irritability and hypermotility proved to exist not only by symptoms but also by x-ray examination it is evident that the diet must be bland and non-stimulating. This will include milk, eggs, well-cooked cereals, purées of vegetables and foods made from these various materials, such as custards, cream soups and rice puddings. Milk may need to be peptonized, if the stools show large curds, or malted milk made from the prepared powder and hot water may agree better than fresh milk, however modified. Eggs should be cooked soft but never given raw. Cereals such as farina, germea or cracked wheat should be cooked at least three hours. Bread should be white, not whole wheat or graham, and agrees best when toasted crisp. Vegetable purées may be eaten alone or with milk in cream soups. No diet list can be given to suit all cases, but with the above as a basis more food or less can be allowed, as

time goes on, according to its effect. Attention must be paid also to food temperature, for cold articles or drinks may increase peristalsis and so cause more diarrhoea. Tea and coffee usually are detrimental because too stimulating to the bowel. Fruits, raw or cooked, salads, fruit juices and lemonades are not proper as a rule for the patient with tuberculous ileocolitis.

(2) *Rest in bed* is most important at the outset of treatment, not only for the sake of decreased bodily activity but also to prevent the sagging of abdominal viscera and their dragging on their supports, such as occurs in the erect posture. Gradually, as bowel movements become less frequent, as pain and tenderness subside, and as the patient gains a little weight and strength, sitting up for a part of each day may be permitted, at first for one hour or two and finally for half of the twenty-four hours. But full activity and exercise are to be forbidden until improvement in bowel function and in general health appear to justify their resumption.

(3) *External applications* over the abdomen probably are the most useful agents of all in the treatment of intestinal tuberculosis. The methods commonly employed include (a) heliotherapy, (b) the quartz lamp, (c) x-ray. (a) *Heliotherapy* is the simplest and most efficacious method of light therapy, and the only limitations to its use are climate and season where there is a minimum of sunshine. The patient should lie with the naked abdomen exposed directly to the sun's rays. At first this exposure should not exceed five to ten minutes each day because of the danger of sunburn. But gradually the duration of treatment can be increased, until finally it amounts to two or three hours each day. Some patients respond better when the entire body is exposed directly to the sun instead of the abdomen only. This can be done in the same way and with

the same precautions as to duration of treatment. Heliotherapy has at least the virtue that it costs nothing and is therefore at the disposal of the poorest patient. But in addition to this, there can be no question about its value in effecting improvement both in the intestinal and in the general condition of the patient. (b) The *quartz lamp* gives an intensely brilliant light, obtained by passing a current of electricity through quicksilver in a quartz tube. If the patient's abdomen or entire body is exposed to this light, much the same beneficial effects are obtained in intestinal tuberculosis as from the sun's rays, and the quartz lamp possesses the advantage that it can be used in all climates and at all seasons. But certain precautions must be observed. The eyes of the patient must be protected by dark glasses, the duration of treatment must be even more limited at the outset than in heliotherapy to avoid burns, and the distance of the lamp from the body must be carefully regulated according to effects. Altogether, therefore, the use of this lamp ought to be under the control of those who understand it and never given to the patient to apply without supervision. (c) The *x-ray* method of treatment must be employed with even greater caution than either of the foregoing, because its effects are not only superficial but deep and may increase the intestinal ulcerations or aggravate them, if used in too large dosage or too frequently. It should be administered, therefore, only by those familiar with its use, but in proper hands it is undoubtedly a valuable aid in the treatment of tuberculous ulcerative ileocolitis.

(4) *Drugs*. — It is extremely doubtful whether in this disease any drugs are of use except for the relief of symptoms. For the pain of acute exacerbations no remedy equals opium, given as the tincture or the camphorated tincture. To check

the profuse or too frequent bowel discharges that usually form a part of the acute exacerbation, bismuth subcarbonate may be added to the therapy. The same prescription as that advised for acute colitis, administered in the same way, is as useful here as there. Sometimes this gives better results if it follows castor oil in dose of one ounce (30 c.c.). If the patient is very weak, and a stimulant is indicated, some alcoholic preparation is preferable such as brandy, or whisky, or port or sherry wine. For food value as well as stimulation panopeptone will be found useful. For foul-smelling discharges, obviously due to secondary invasion of the bowel by saprophytes, creosote is indicated, two minims (0.13 c.c.) in capsule once in four hours, or one tablespoonful of liquid peptonoids with creosote; five grains (0.3 gm.) of salol may be given once in four hours, instead of creosote. The usefulness of the various tannic acid preparations is dubious. Finally there is no specific remedy for tuberculosis in the intestine any more reliable than for this disease in the lung.

(5) *Surgery*. — Limited areas of tuberculous ulceration in the bowel can be excised successfully, as for instance, the tuberculous cæcum. Localized intestinal tuberculosis leading to stenosis and obstruction may require surgery as an emergency measure regardless of ultimate cure, and the same is true of perforation by a tuberculous ulcer. In certain cases also good results have been obtained by appendicostomy or colostomy for purposes of irrigation of the bowel. But the opportunities for benefit from surgical interference are rare, the mortality from operation is high, and authorities agree that it is not often that this method of treatment is justifiable.

CHAPTER XVIII

CHRONIC ULCERATIVE COLITIS: NON-SPECIFIC

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After *Entamæba histolytica*, Shiga bacilli and tubercle bacilli have been excluded as possible causes of a chronic ulcerative colitis, there remains a group called non-specific or idiopathic for which no organism has so far been generally accepted as responsible. At one time during the past decade it was thought that this group might be due to a specific diplococcus described by Bargaen. He found this in the secretion covering the multiple miliary lesions in the lower bowel, as well as in the stools, was able to culture it outside the body and to reproduce the characteristic lesions in the colon of rabbits and dogs by injecting intravenously cultures of this diplostreptococcus. Furthermore, he was able by means of an antiserum made by immunizing horses to the diplostreptococcus to benefit a large number of the patients seen. Other workers in other clinics, however, were unable to identify the organism described by Bargaen, and considerable doubt was thus thrown upon its specificity. At the present time Bargaen still expresses his confidence that the diplostreptococcus is the specific cause of the disease and states that he finds it in 80 per cent. of the patients examined. He continues to advise the use of the anti-serum and claims that in the other 20 per cent. of ulcerative

colitis cases, in which he failed to find the organism, the results of treatment by the serum are as good as in those where it has been found.

Whatever the cause, this form of chronic ulcerative colitis is a most serious one and has always proved intractable to all kinds of medical treatment. Usually it begins in the rectum, spreads from there throughout the colon and even to the terminal ileum. It involves not only the mucous membrane but ultimately all the layers of intestinal wall. It causes much suffering, incapacity for work and general debility, even though it does not quickly lead to fatal outcome. Fortunately it is not a condition frequently seen, and the experience of no one physician or hospital is large. Bergen, however, bases his conclusions upon the observation of over 1500 cases referred to the Mayo Clinic in the past ten years, and in certain localities it is seen more frequently than in others.

### CLINICAL HISTORY

The symptoms produced by this form of chronic ulcerative colitis are not essentially different in character from those seen in other groups, but usually they are more severe and less subject to remission. Increased frequency of bowel discharges, bloody, mucopurulent stools, pain, tenesmus and straining, gradual loss of color, weight and strength, with partial remission at times for weeks but rarely complete restoration to normal even during these periods of lessened severity and a duration for months or for years are the characteristic features.

### EXAMINATIONS

*Physical examination* shows only a tender abdomen but no distinctive signs. The *proctoscope* reveals a granular, reddened

mucosa with multiple miliary ulcers in the lower bowel that bleed easily. This picture of a widespread hyperæmia and œdema of the bowel surface, easily irritated by instrumental interference to the point of discharging blood, is unlike any other seen in chronic ulcerative colitis. The *stools* on gross inspection are soft, liquid or semi-solid and consist largely of mucus, pus and blood with which the fæcal material is mixed. As already noted, there is a difference of opinion as to whether any specific infective agent can be found by cultures, but all agree that this form of intestinal disease is not due to amoebæ, Shiga bacilli or the acid-fast organisms of tuberculosis. Even those who admit that the diplostreptococcus described by Bargaen can be identified in cultures claim that it is simply one of the pleomorphic streptococci and not to be looked upon as specific. *X-ray examination*, as in any form of severe colitis, shows after a barium enema absence of haustrations and narrowing of the lumen, particularly in the sigmoid and descending colon, but no sign diagnostic of this form as distinguished from others.

### TREATMENT

This must first of all be the same in general as that advised for any form of severe chronic ulcerative colitis, by a soft, bland diet, of high calory value and with little residue; by rest in bed at least half of the twenty-four hours and constantly, if the exacerbation is severe, and by opium sufficient to relieve pain and control excessive bowel discharges. In this group of cases the pathological lesion has its site low enough in the bowel to justify the use of cleansing and soothing colonic flushes with warm normal salt or weak bicarbonate solutions once a day or every other day. But these are intended only

for relief of symptoms. Solutions of antiseptic substances, intended for cure, not only fail of their purpose because the disease extends beneath the surface of the bowel beyond their influence, but also sometimes produce irritation that does more harm than good. Different methods of treatment must be employed, if the object is to rid the bowel of infection. The disease is not a common one, and few physicians either in private practice or in hospital service have opportunity to see so large a number of cases as gives the right to speak with authority about the kind of treatment preferred. But definite views have been expressed by several, as a result of large experience, about the value of two different biologic products in treating chronic ulcerative colitis.

*First*, based upon the observation of over 1500 patients, Bargaen advises the use of a serum made from horses immunized by cultures of a diplostreptococcus obtained directly from the intestinal lesions of patients suffering from chronic ulcerative colitis. This preparation is now available for general use under the name "ulcerative colitis antistreptococcic serum". It is to be given intramuscularly, by choice in the outer area of the thigh, or else intravenously. The dose is 1 c.c. gradually increased to 5 c.c., given daily or every other day, as the patient's condition indicates. Bargaen states that 75 per cent. of the patients treated in this way become clinically well, responding promptly by a fall in temperature, lessening of the symptoms of intoxication and decrease in the number of stools.

*Second*, Hurst of Guy's Hospital, London, called attention over ten years ago to the value in this form of chronic ulcerative colitis of a polyvalent antibacillary serum made from different strains of the Shiga-Flexner organism on the theory that this disease is caused originally by bacillary infection, even though

these organisms have gradually disappeared from the bowel because overgrown by strains of streptococci. This plan of treatment has been employed by Crohn (*Crohn and Rosenak: Am. Jour. Digest. Diseases*, August 1935, p. 343.) at the Mt. Sinai Hospital in New York, and he reports upon his results in 90 cases, observed from two to fourteen years. Of this group 75 per cent. were cured or improved. He gave every other day 5 to 20 c.c. of the polyvalent serum intravenously, the size of the dose depending upon the degree of reaction.

Most observers seem to feel that no matter what serum is employed, the beneficial effects are non-specific and the result of protein shock, and that by producing this, no matter whether polyvalent antibacillary, or Bagen antistreptococcic or typhoid or even plain horse serum is used, the patient's condition gradually improves. When serum sickness with urticaria and joint pains follow, the results are even better as regards the bowel condition, because these symptoms indicate that definite protein shock has been produced. Crohn is of the opinion, however, that while all types of intravenous serum therapy are beneficial in this way and not because of any specific effect on the disease, the use of polyvalent antibacillary serum gave better results than any other.

In case all methods of medical treatment fail, surgery has to be called upon to aid. The procedure indicated then is an appendicostomy, cæcostomy or ileostomy, not only to give rest to the colon but also to permit irrigation of it from above downwards with medicated and slightly antiseptic solutions. For this purpose the remedies commonly employed are acroflavine hydrochloride in 1 to 4000 solution or mercurochrome one or two per cent. The bowel drainage and irrigation must be continued for a long time, at least one year and possibly

two, before any attempt is made at restoration of normal continuity by closing the drainage wound. Restoration after colostomy or ileostomy not infrequently, however, is fatal; if not, often all symptoms quickly reappear.

The failure to cure by any methods of treatment ordinarily employed are shown by the following case history, which is presented in some detail, because it illustrates the tendency of the disease to remission and exacerbations, response temporarily to treatment, followed by relapse and the discouraging recurrence of symptoms sooner or later after every effort to remove the intestinal ulceration which causes them.

A man, aged 27, first came to the writer for advice in December 1927. His story was that in the preceding May he began to have dysentery with cramps in the lower abdomen, persisting during May, June, July and August. In August he had entered Mt. Zion Hospital and remained there six weeks. He left then because so much improved but soon had to return because of recurrence of his symptoms and remained this time until October 30th. His condition again improved during his stay but never became normal. After he left, he continued to have stools with no form at all, almost like water, "passed with a big splatter", and in general, when seen in December, he said he felt worse than at any time since leaving Mt. Zion at the end of October. The patient was pale but fairly well nourished, and physical examination showed no evidence of organic disease except tenderness and moderate distension of the abdomen. The stools were soft and thin, light, brown, showed little mucus, no visible blood but definite reaction for occult blood, no amoebæ or cysts. There was no abnormality of gastric secretion shown by gastric analysis.

This patient was kept under observation at intervals during the first half of 1928, while he continued at work, up and about. He improved at first on a non-residue smooth diet, more rest and less activity. His bowels on this plan moved only once or twice a day,

and the stools were more mushy than liquid. But within a few months his griping pains recurred, his bowel movements again became loose and frequent, he lost weight, strength and color and could not do his work. He was then advised to enter the Medical Clinical Ward at Stanford Hospital for more complete investigation than could be made while he was ambulatory. He did so in July 1928.

In the hospital proctoscopic examination showed there were numerous small ulcers in the ampulla and sigmoid, varying in size from 2 to 4 mm., covered with gray exudate and bleeding easily when this was removed. Smears from these ulcers showed an occasional red blood corpuscle, many pus cells, large Gram-positive bacilli, slender Gram-positive bacilli, large Gram-positive cocci in pairs, small Gram-positive cocci in pairs, small Gram-positive cocci in long chains and Gram-negative bacilli. No acid-fast bacilli were seen. Cultures on special media were negative for bacteria of the typhoid, paratyphoid and dysentery groups. Cultures showed growth of non-hemolytic streptococci which failed to grow on sub-culture and could not be identified. Blood cultures were negative. X-ray of the gastrointestinal tract showed no disease except hypermotility of the colon.

On admission to the Stanford Medical Clinic Ward the patient was found to have a high septic type of fever that persisted for two weeks, gradually falling during another two weeks, before it became normal. There was continuous diarrhoea, the movements averaging five to eight each day with cramps. An autogenous vaccine was prepared from his stool culture and given every few days during July and August, and daily irrigations of the lower bowel were made with warm normal salt solution. On this plan he gradually improved as regards his fever and his diarrhoea but not in the condition of his lower bowel as shown by the proctoscope. After his dismissal from the ward in August 1928 he continued to return to the outpatient clinic for his vaccine injection during the remainder of 1928. But the diarrhoea persisted, better or worse, with bowel movements never normal, and proctoscopic examination in December 1928 showed no improvement in the ulcerated area.

This patient was not seen again until June 1931, when he returned to Stanford Medical Clinic for further care. He reported that early in 1929 he was treated for six weeks at another hospital in San Francisco by enemas of neutral acroflavine in normal salt solution, which he said "completely cured him", but within a month after discontinuing his enemas, the trouble recurred. Then he went to Chicago, and while there, had his gallbladder removed in May 1929, because told that disease of this organ caused his colitis. He was much improved again as regards bowel symptoms after this operation, but not for long, though never ill enough to require hospital care after that until his report here in person in June 1931. He complained then of cramps and diarrhoea as usual, and the proctoscope again showed the sigmoid oedematous, reddened and covered by numerous pin-point ulcers that bled easily. Under treatment he improved again and remained fairly well for some time.

His next entry to the Medical Clinic Ward was in March 1932, when he told the same old story of diarrhoea and cramps. He had remained fairly well since his previous treatment a year before but never normal. He showed no new signs on physical examination, but his abdomen was tender and distended. The laboratory examination of stool revealed no different findings on microscopical examination of smears or in cultures from those at the first entry in 1928. X-ray films of the colon after a barium enema now showed the descending colon very definitely ulcerated, haustrations being smoothed out by the infiltrating processes. The proctoscope showed the same ulcerations as formerly. On a high caloric, high vitamin, non-residue diet, bed rest, daily colonic irrigations with one per cent. solution of soda bicarbonate, the patient improved rapidly. But one month after dismissal, when he returned to work, he began again to have pain and many loose stools each day, in spite of continuing all treatment except rest.

After this the patient returned to Chicago and has not been seen since, but in March 1933 he reported by mail that in December 1932 he consulted a physician in Chicago, who gave special attention to

ulcerative colitis and had been treating him since by a polyvalent vaccine with considerable improvement. But apparently this did not have any more permanent good results than other methods of therapy previously employed, for his last communication, in May 1934, said that he was still troubled by the same symptoms. It should be emphasized that repeatedly and by a number of different physicians and surgeons, who saw this patient at various times, he was advised to have an ileostomy or cæcostomy, but would never consent.

## CHAPTER XIX

# LOCALIZED INFLAMMATION OF THE INTESTINES: REGIONAL ILEITIS

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There are certain localities in the course of the intestine that seem particularly subject to attack by infection. In these the micro-organisms responsible are not always identified and are not always the same. The symptoms vary according to the region involved, and the designation for each is given according to its site. The principal areas thus affected are the ileum, the appendix and the sigmoid colon, and the syndromes that result are known as regional ileitis, appendicitis and diverticulitis.

The name regional ileitis was suggested by Bargaen, but recognition of the disease and first description of it as an entity was by Crohn. It is a comparatively recent addition to the number of intestinal diseases, probably because it was previously overlooked, for it was first definitely given a place in medical literature in 1932. In the beginning it was thought to involve only the terminal ileum, and this is still correct in most cases, for the last eight or ten inches of this portion of the small intestine are the usual site of the pathological lesion. But observations since the first accounts by Crohn himself and by others, who have published case reports, have widened the

conception of the area that may be affected. It is now recognized that the entire ileum may, exceptionally, be the seat of pathological change, and at times this may extend even into the jejunum. The fact remains, however, that the lower foot of the ileum, instead of the entire twelve feet of its length, is the part of the bowel particularly concerned in so-called "regional ileitis", but Koster, Kasman and Sheinfeld, *Archives of Surgery*, May 1936, p. 789, report similar cases observed that involved the large bowel beyond the cæcum, and think the disease may occur in any part of the intestinal tract.

It is not a common disease, but Crohn has accumulated in three years the records of between thirty and forty such cases. Other clinics and individual observers have reported a few instances here and there from widely separated points in this country, but the frequency noted at Mt. Sinai Hospital suggests that Hebrews may be more subject to it than other races.

#### ETIOLOGY AND PATHOLOGY

The etiology of the disease is not known. No micro-organisms that might be considered causative have ever been found in the bowel wall examined at once after its surgical removal. It has been suggested, however, that the Shiga bacillus or one of its strains may be the agent responsible and that agglutination tests should be made in all cases. The pathological changes observed in the ileum are described as due to a chronic, proliferative inflammation with ulceration of the mucosa, thickening of the wall, narrowing of the lumen, constriction and stenosis. These changes may extend a variable distance upwards in the ileum, but downwards they stop suddenly at the ileocæcal valve and are not found beyond it.

Perforation of the bowel wall is apt to occur with the formation of fistulæ between the diseased ileum and the colon or the external wall of the abdomen. Lymph nodes in the mesentery adjacent to the bowel involved usually are found enlarged.

### CLINICAL HISTORY

The symptoms produced by this disease are cramps of greater or less severity, more or less constant diarrhoea, variable fever but rarely high, discomfort and tenderness in the right lower abdominal quadrant and gradual loss of weight and strength. This may go on for months or years, better and worse, before it is recognized. A natural conclusion from such a history is that the patient has a chronic appendicitis, and it is not uncommon to hear as a part of the story that the appendix has been removed without influencing the symptoms. Or the clinical picture may be misinterpreted to mean ileocæcal tuberculosis with unsuccessful attempts at alleviation by light therapy, diet and the usual hygienic measures employed in such cases for a variable period before the conclusion is finally reached that there must be some other explanation so far overlooked.

### EXAMINATIONS

*Physical examination* usually is negative except for tenderness over the right side of the abdomen in its lower half. The wall of the ileum, however, exceptionally may be sufficiently thickened by chronic inflammation to be palpable through the abdominal wall as a tender ill-defined mass. The *stools* give no diagnostic information by gross inspection, microscopical or cultural tests, and proctoscopy is of course useless, because the pathological changes are far beyond the instrument's reach.

*X-ray Examination.* — Visualization of the terminal ileum by such amount of barium as enters it on its way down from the stomach or by such as may escape upward past the ileo-cæcal valve after a barium enema proves that in this disease the bowel is small, contracted, narrowed and stiffened, irregular and deformed. X-ray films appear to be the best and most reliable aid in diagnosis, but the difficulty about visualizing this part of the bowel makes this method of diagnosis uncertain, and repeated trials sometimes are necessary before success is attained.

### TREATMENT

Even though exact diagnosis is undetermined in advance, and the patient's condition has wrongly been considered chronic appendicitis or ileocæcal tuberculosis or a neoplasm, sooner or later operation becomes necessary, because other measures fail to cure. Then the abnormal thickening of the ileum should be discovered, for now that the possibility of regional ileitis as a cause of the symptoms is recognized, the ileum should always be carefully inspected and palpated at the time the abdomen is opened. The only cure lies in resection of the diseased portion of the bowel with subsequent anastomosis between its cut end and the colon. The results of this operation reported in medical literature have been quite uniformly successful in restoring the patient's health.

A man, aged 27, was referred to the Surgical Clinic Ward at Stanford Hospital in September 1934 for treatment of a sinus and fæcal fistula through his abdominal wall ever since an appendectomy in 1932. Since the fifth day after appendectomy he had a tiny opening at the base of the operative wound, draining a thin greyish-brown discharge. Appendectomy had been performed

because he awoke early one morning with severe abdominal pain, nausea and vomiting. Operation was done the same day. In the surgical clinic ward physical examination was negative except that a thickened mass could be felt around the sinus. A probe could be passed through the fistulous opening for an inch and a half to two inches. After injection of the sinus with lipiodol, x-ray films showed that the tract led to the cæcum, and the lipiodol could be seen later on in the ascending and transverse colon. Cultures from the sinus discharge showed growth of colon bacilli and streptococcus viridans; smears revealed no actinomycosis, blastomyces or tubercle bacilli. Blood count was normal, urine and blood Wassermann negative. At operation the terminal ileum was found markedly thickened for the last 20 cm. of its course to the extent that it formed a fusiform, tumor-like swelling of the ileum approximately 4 to 5 cm. in diameter. The surface was reddened, the mesentery thickened and several large firm glands could be felt in it. The tumor was excised and the terminal 18 to 20 cm. of the ileum together with the cæcum. A side to side anastomosis was made of the ileum to the colon. Microscopical examination of the specimen removed showed hyperplastic mucosa heavily infiltrated with small round cells and occasional polymorphonuclears, marked thickening of the submucosa and muscularis with increased connective tissue and no evidence of tuberculosis or malignancy. The patient made a prompt recovery after operation and has remained well since.

## CHAPTER XX

### LOCALIZED INFLAMMATION OF THE INTESTINES (CON'T.)

#### APPENDICITIS

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The appendix is the site in the intestines most subject of all to infection and consequent localized inflammation; it is much more often attacked than the ileum, where regional ileitis by comparison is rare, or even than diverticula in the sigmoid, where resulting diverticulitis resembles it in its clinical course but not in the locality where its manifestations are produced.

#### ACUTE APPENDICITIS

Acute appendicitis is one of the most serious diseases that ever involves the intestinal tract, and though it starts in a small area, it rapidly spreads beyond its point of beginning, and frequently proves fatal unless recognized promptly and attacked vigorously without procrastination. Any acute painful attack in the abdomen may prove to be due to appendicitis. Delay in realizing what the symptoms mean and instituting correct

treatment for their cause is the common explanation of a fatal outcome. Hence the importance of keeping constantly in mind this type of localized inflammation of the intestine and of always being on the lookout for it.

### CLINICAL HISTORY

So much has been written in recent years about this subject, not only in medical journals but also in newspapers and popular magazines, that patients as well as physicians have learned to know how acute appendicitis makes its onset and how rapidly it may progress unless its course is checked. The characteristic features in the beginning are pain, tenderness, vomiting and fever. Suddenly, by night or by day, there comes the consciousness of pain in the abdomen. At first this may be slight and widespread, but soon it grows more severe and more localized with its greatest intensity in the right lower quadrant. The pain may be colicky in character, intermittent, increasing and decreasing but never entirely disappearing, or it may be a dull, steady ache that grows continually worse and worse with a sense of distension as if something inside would burst. With this comes tenderness and soreness over the area of pain, of which the patient is well aware before the physician finds it at his examination. Vomiting is apt to be an early incident, or at least nausea and the feeling that emptying the stomach will relieve the pain. But vomiting does not give relief or prevent the progress of the attack. The bowels may move once at the outset emptying the rectum, but this likewise does not stop the pain, nor does an enema; a physic should never be given, no matter how strongly the patient feels that this will remove all the trouble. Fever is found when the thermometer is used, but usually it is low at the outset and not enough to make the

patient aware of its presence. Increased pulse rate usually accompanies the rise in temperature.

The subsequent course is variable. No one can tell in the beginning how virulent is the infection or how far the pathological change has advanced or what the developments will be. There are several possible outcomes of an attack. *First*, after two or three days of moderately severe symptoms, either spontaneously or in response to therapy, the pain subsides, the tenderness disappears, and the temperature falls to normal. This is the most favorable course possible. Attacks of this sort may recur a number of times at variable intervals before one appears that is so entirely different it quickly ends life. It is much wiser, warned by one or two of these comparatively trivial experiences, not to wait for a more severe one before proceeding to appendectomy. *Second*, instead of the pain gradually disappearing after twenty-four to forty-eight hours, it increases in intensity or remains constant with tenderness likewise persisting and temperature even rising higher. Even so, with this more violent degree of infection, the attack may ultimately subside after a week or ten days without leading to any disaster. But the risk in such cases is great, and the outcome so uncertain that delay is never justified and should not be advised. *Third*, after a few hours or one day of suffering, sudden cessation of pain may occur and the patient feel so greatly relieved that he concludes the attack is over. But always this sudden change for the better is treacherous and not to be trusted. It usually means that the appendix has ruptured, its contents have escaped, and thus the tension that caused the pain has been overcome. But the temperature does not fall, the pulse rate does not return to normal, and sooner or later the former discomfort returns. *Fourth*, after rupture of the

appendix, the purulent contents extruded usually find organized adhesions around the site of inflammation that limit its progress and wall it in so that a localized abscess results with localized peritonitis about it. Then pain, tenderness and persistent fever give warning of what has occurred, and physical examination and laboratory tests identify the nature of the trouble. But sometimes the infection is so virulent, the progress of the inflammation so rapid, and the contents of the appendix escape so early, that not sufficiently strong adhesions have been formed to restrain them. Then they find their way directly into the peritoneal cavity, and the result is acute general peritonitis. This is a possibility threatened in every instance, no matter how simple the attack seems at the outset, and hence the necessity for early diagnosis, close observation of the course of events and resort to surgery without too great delay.

The clinical history, however, offers a number of variations from the typical one just described, and these sometimes confuse the picture. These variations concern the site, the character and the radiation of the pain, the kind and degree of disturbance of gastrointestinal functions produced by the attack and the fever course. (1) The *pain* may be felt with greatest intensity as high in the abdomen as the level of the navel or even higher, in the right hypochondrium when the appendix is situated behind the cæcum with its tip pointing upwards behind the ascending colon, or not in front at all, but behind in the right loin. In some instances the site of pain may be the epigastrium or even the left hypochondrium. But these unusual situations of the pain are, as a rule, noted only in the beginning, and within a few hours it settles down in the right lower quadrant and remains there subsequently. The character

of the pain is not always the same. Most often it is like colic at first with cramps coming and going across the lower abdomen, with comparative freedom from all pain between them, but ultimately, as the site becomes restricted to the appendix area, the suffering becomes more constant and less intermittent. From the outset, however, the pain may be a dull, steady ache without relief, gradually increasing in degree to an almost intolerable sense of fulness. A most deceptive and treacherous form of the disease is that with pain so slight it seems the pathological lesion causing it must be of little consequence, until finally rupture of the appendix takes place suddenly and unexpectedly. Such cases usually are the result of gangrene of the appendix, due to shutting off of its blood supply by torsion or pressure, without excessive distension of its lumen by the products of inflammation. Finally, while the greatest suffering usually is experienced in the right lower quadrant of the abdomen, the pain may radiate downwards into the scrotum and right testicle in the male or to the perineum and inner side of the right thigh in either sex, or upward along the right side of the abdomen to the right costal margin, or into the right back. All these unusual paths of radiation may lead to misunderstanding as to the site of the pathology causing the pain, unless it is noted that the site of maximum intensity is in the right lower quadrant, as almost invariably occurs in acute appendicitis.

(2) Variations in the kind and degree of *gastrointestinal disturbance* caused by acute appendicitis are frequent, and these also may mislead. Nausea and vomiting may be so persistent and severe at the outset that it seems certain that the lesion causing this is situated in the stomach, while on the other hand these symptoms may occur only once or twice as the

attack begins and not at all afterwards. The intestines similarly may be at times the apparent site of the acute disease with cramps, diarrhoea and all the other evidences of an acute colitis. But as a rule there is no more than one bowel movement in the beginning and none thereafter. An enema does not give relief, and if the patient takes a cathartic, it only increases his pain. Thus acute appendicitis may simulate at the outset either acute gastritis or acute colitis, but should never deceive, if the possibility that the symptoms may mean acute appendicitis is kept in mind in every acute abdominal attack.

(3) *Fever* is not high in acute appendicitis unless some complication causes it. So trustworthy is this rule, that if high fever is found in the beginning it suggests some other disease as a cause. Gradual rise of the temperature first observed, from a point below  $100^{\circ}$  to a point between  $101^{\circ}$  and  $102^{\circ}$  F. means progress of the inflammation. Similarly a gradual fall means that the inflammation is subsiding. Sudden fall is ominous because unnatural and usually is the immediate effect of perforation or rupture. Rise in temperature after it has once fallen means that some complication has occurred, such as abscess or localized or general peritonitis. The most deceptive cases from the standpoint of fever are those that show none at all or only a slight rise, below  $100^{\circ}$  F. in spite of rapid involvement of the appendix and its rupture even before realization that it is seriously diseased. Such fulminant cases are infrequent, but when they occur, the temperature is not a trustworthy guide.

Thus it becomes evident that there is no single fixed pattern that this disease always follows, and that variations, such as described as well as complications that may result, may cause

a wide divergence from the average clinic course. Symptoms alone never explain what is going on, and physical signs, temperature course and blood counts are also essential for a correct interpretation of the sequel of events.

### PHYSICAL EXAMINATION

The characteristic signs of an acute appendicitis add much to the evidence already furnished by the symptoms. These signs are fulness, tenderness and rigidity found in the right lower abdominal quadrant. The fulness appears early but tenderness comes even sooner. The swelling may be perceived by inspection as a plumping up of the wall over the right iliac fossa so that the normal iliac groove is obliterated or much less distinct on the right side as compared with the left; or it may be found only by palpation, the finger tips detecting what the eyes do not yet see. The tenderness is often discovered by the patient even before the physician makes his examination, for as a rule it appears with the pain. The diagnostic point of greatest tenderness is about half way or two-thirds of the way out on a line drawn from the navel to the right anterior superior spine of the ilium. But this site is not constant. It may be found farther out toward the iliac crest, or farther in toward the navel, or lower down toward Poupart's ligament, or higher up on a level with the navel or even a little above it. These variations all occur, and any one of them is fairly typical. But the area within which tenderness is found is almost invariably limited to the right lower quadrant, and if it is situated elsewhere in the abdomen, probably the pathological lesion is not in the appendix. The importance of determining this site lies in the fact that the pain at the outset may be referred to some point far away, such as the epigastrium, or the right costal

margin or the right loin, but if the most sensitive spot on palpation nevertheless lies in the right lower quadrant, the pain can be safely assumed to originate in disease of the appendix and not in that of some other organ. Even when the first pains are felt all over the lower abdomen and the tenderness also is widespread, still the maximum complaint is elicited when pressure is made over the appendix area. Rigidity over the same region as the fulness and the tenderness is due to muscle spasm and is protective. It not infrequently is found on the right side in marked contrast to the corresponding area on the left, even when tenderness is not produced by pressure, because the presence of muscle spasm purposely prevents deep palpation. When an acute painful attack involves the abdomen, these three signs, swelling, tenderness and rigidity over the right lower quadrant, are highly trustworthy and almost but not completely diagnostic.

The signs that indicate an abscess has formed around the appendix, whether or not the appendix has ruptured first, are a definite, localized, palpable mass, differing in consistence from the surrounding parts, as large as a hen's egg or sometimes as large as an orange, tender to pressure, with decreased resonance over it as compared with the tympany of the adjacent bowel. The temperature course after this unfortunate complication has developed tends to become septic in type, normal in the morning or nearly so, about  $101^{\circ}$  F. or  $102^{\circ}$  F. in the evening, and the pulse correspondingly is quickened as in septic fever from any source. When acute general peritonitis follows any of the preceding developments, the tenderness, previously localized, becomes wide-spread and extreme. The whole abdomen, instead of a small well-defined area, develops rigidity, and general distension rapidly takes place. The fever rises to a new high

level, 103° F. to 105° F., and the pulse becomes not only extremely rapid but small in volume and thready in tone.

### BLOOD EXAMINATION

To the evidence supplied by history and by examination of the abdomen a very valuable addition is made by counting the white blood corpuscles. Very early in its course acute appendicitis causes an increase in the number of these cells. As the inflammation progresses, the number of leucocytes rises, so that a count at the outset and again two hours later tells not only whether the inflammation is advancing but the rapidity and degree of its progress. This becomes, therefore, as valuable a diagnostic guide as pain, tenderness, fever and the physical signs described. The average case presents a leucocytosis of twelve to fifteen thousand. When an abscess has formed, this may increase to twenty thousand or above. With acute general peritonitis the count is usually between twenty and thirty thousand. The case that makes its onset without leucocytosis, with a normal or decreased number of white cells, is more to be feared than one that responds in the usual way, for it means either a virulent infection or a poor reaction on the patient's part and in either case a dubious outcome.

### DIAGNOSIS

Sudden pain in the right lower abdomen, nausea and vomiting, not much diarrhoea in spite of the cramps, rather a tendency to constipation, tenderness, swelling and rigidity at the site of pain, moderate fever and acceleration of pulse, increase in the leucocyte count, these are the pieces of the puzzle. Fit them together, and the picture they make is called acute appendicitis. As a rule this picture is perfectly clear, but sometimes

the color of the pieces is dim, or their pattern is blurred, so that the composite whole when they are combined is indistinct.

*Acute Gastroenteritis.* — When acute appendicitis begins, as it often does, with general rather than local disturbances in the abdomen, the first thought naturally is of one of the everyday illnesses such as acute gastritis, enteritis or colitis. If vomiting initiates the attack and is repeated frequently, thus assuming the chief rôle, with discomfort across the upper abdomen, with slight fever and leucocytosis, the reasonable inference is that the stomach is the seat of the disease. But when gradually discomfort develops into more definite pain, and its site of maximum intensity shifts from epigastrium to right lower quadrant, when the well-known signs of tenderness, distension and rigidity appear over the appendix area, when fever persists and grows higher, pulse rate rises and leucocytes increase steadily in number, then it usually becomes apparent, within a few hours, that the appendix and not the stomach is the part involved.

So when the attack begins suddenly with cramps and diarrhœa, discomfort and soreness over the lower abdomen, fever of low grade and moderate leucocytosis, the inclination of the physician is to consider the illness trivial, due to a transient inflammation of the bowel that will soon respond to simple therapy. But if the pain and tenderness gradually limit themselves to the region of the appendix and remain there, if diarrhœa ceases after a few discharges, if the temperature tends to increase and not to disappear, the pulse grows quicker and the leucocyte count rises, if swelling and muscle spasm as well as tenderness make their appearance over the right lower quadrant, it then should become clear, if the physician keeps close watch, that the illness is not so unimportant as it seemed at first to be.

*Cholecystitis.* — If from the beginning the evidence points to

some acute disorder in the right side of the abdomen, but without clearly fixing its site, it then becomes necessary to consider all the other organs besides the appendix that are subject to such acute attacks. First in importance among these comes the gallbladder, which is almost as common a site for acute inflammation as the appendix itself. But the clinical picture of acute cholecystitis usually is sufficiently different to leave no doubt between the two. The pain is most intense at the right costal margin. It radiates to the back under the right shoulder blade, or into the epigastrium, or upwards under the sternum, but occasionally downwards on the right side and then may cause confusion with the pain of appendicitis. But even so the tenderness and sense of fulness, the rigidity and spasticity over the right lower quadrant that characterize appendicitis no matter where the pain radiates, are not found there now, but instead are discovered in the right upper quadrant, below the edge of the ribs. The temperature is moderately elevated, the pulse rate quickened, and leucocytes increased in one condition as in the other. Sometimes, it must be admitted, the picture is not clear, and differentiation is impossible for the reason that the gallbladder and appendix are involved simultaneously. But more often each preserves its own features with sufficient distinctness to permit an accurate conclusion to be reached.

*Pyelitis.* — Acute inflammation originating in the pelvis of the right kidney likewise makes a sudden painful onset. But here a chill is much more commonly the prelude. Chill does not often mark the onset of appendicitis, but it is not so rare that its occurrence precludes the possibility. The pain of acute pyelitis is felt first in the loin, around toward the back. But so it may be in acute appendicitis when inflammation attacks a retrocæcal appendix that lies with its tip upward behind the

ascending colon. The radiation of pain in pyelitis is downwards and inwards toward the bladder and genitalia with disturbance of urinary function, with increased frequency of urination, more or less constant desire to void, and the discharge of urine that is turbid and sometimes bloody. But even these signs of irritation in the urinary tract may be produced at times by acute appendicitis, except the visible changes in the urine. The maximum tenderness in pyelitis is elicited in the back at the right costo-vertebral angle, and the diagnostic signs of appendicitis are not found in the right lower quadrant. The temperature is elevated in each. But fever usually is higher at onset in pyelitis and may be  $103^{\circ}$  F. or  $104^{\circ}$  F., when the patient is first examined, a point always against acute appendicitis. Both conditions produce a moderate leucocytosis, but pus and blood in the urine in definite amounts are not seen in appendicitis and are characteristic of pyelitis. Cases have been observed where red blood cells were discovered in the urine early in an attack of acute appendicitis with no evidence otherwise of kidney disease, but only in moderate number and by the aid of the microscope. Probably they are due to congestion of the right ureter caused by the adjacent inflammation of the appendix. But this is the exception, not the rule.

*Salpingitis.* — In women acute inflammation of the right Fallopian tube may closely resemble acute appendicitis. The sudden onset with pain in the right lower abdomen and with fever and leucocytosis inevitably is delusive. But the pain in salpingitis is lower, and when it radiates, it does so down the inner side of the thigh. The most important point in differentiation is the fact that the maximum tenderness is found by vaginal examination in the right broad ligament with brawny induration of the tissues there and fixation of the uterus, while the usual

diagnostic signs over the appendix area are missing. But sometimes when the cæcum is low in the abdomen, the appendix may hang at the pelvic brim, and when it is inflamed, may be palpated by vagina more readily than through the abdominal wall. This abnormal arrangement easily may lead to confusion, but it does not often occur. Usually the exquisite tenderness discovered by vaginal palpation to the right of the uterus, while little or none is found higher up in the abdomen over the usual site when the appendix is inflamed, can be accepted as positive proof of acute salpingitis. Both tube and appendix are sometimes involved together, when the latter's position is low, so that at operation one as well as the other has to be removed.

*Gallstones.* — There are other acute painful attacks in the right side of the abdomen, not inflammatory but mechanical in origin, yet resembling closely in many details acute appendicitis. These likewise involve the gallbladder, the right kidney, or the right broad ligament. The typical gallbladder attack is that produced when a gallstone attempts to escape through the ducts to the bowel. It resembles acute cholecystitis as regards the place, the character and the radiation of the pain, and the site and nature of the physical signs. But there is no fever nor leucocytosis as a rule, and obstructive jaundice is more likely to accompany or follow.

*Renal Colic.* — The typical kidney attack is renal colic, due to the passage of a stone down the ureter. Here again the site of pain, the disturbances of urinary function, the presence of blood in the urine and the tenderness at the costo-vertebral angle are exactly as described in acute pyelitis, but there is usually no fever and no increase in leucocytes unless stone and pyelitis exist together. A second type of renal attack is that called Dietl's crisis. This occurs most often in women, results

from a movable kidney and is due to the kinking of a ureter with obstruction to the urine and consequent over-distension of the kidney pelvis. Such an attack comes on with sudden pain and with greatest frequency on the right side because the right kidney is the one most often prolapsed and "floating". But the pain is felt higher up and further back than is usually the case in appendicitis; there is no fever or leucocytosis unless the urine is infected; tenderness is found at the costo-vertebral angle, not over the right lower quadrant; a palpable tumor, elastic in character, is frequently discovered below the costal margin, caused by the dilated kidney; the attack terminates with the passage of a large amount of urine when the kink straightens out and ceases to obstruct. Still a third type of painful attack of renal origin is that caused by stricture of the right ureter. This produces more or less constant dull pain in the right lower abdomen with recurring acute exacerbations, but without fever or leucocytosis. Only by cystoscopy and ureteral catheterization after the attack is over can this diagnosis be positively made.

*Ectopic Pregnancy and Twisted Pedicle of an Ovarian Cyst.* — Finally, other pelvic conditions in the right broad ligament, not due to inflammation, may resemble acute appendicitis, particularly ruptured ectopic pregnancy and twisted pedicle of an ovarian cyst. The sudden and violent agony caused by the rupture of a tubal pregnancy in a woman previously healthy, with the history of no unusual preceding symptoms except for a menstrual period missed or delayed, is always a startling and tragic event. The pain is felt low down in the abdomen, and if on the right side, easily may be mistaken for acute appendicitis. But certain features serve to distinguish one from the other. In a ruptured tubal pregnancy the suffering is exceedingly severe even at onset, not gradual in its devel-

opment, and may at the very beginning be accompanied by shock so great that it causes the patient to faint. Pallor quickly results from blood loss with rapid small pulse, but there is no fever. By vaginal examination a tender mass is found in the right broad ligament and Douglass' pouch. Usually there is some bloody uterine discharge soon after the attack begins. All the evidence of pelvic disease thus is so definite that there is rarely any doubt between that diagnosis and acute appendicitis. But serious doubt may arise about the particular kind of pelvic disease present, and to settle this point usually it is wiser to ask the assistance of a gynecological consultant.

Torsion of the pedicle of an ovarian cyst on the right side likewise manifests itself by pain in the lower abdomen varying in degree according to the suddenness with which the torsion takes place. It may be rapid in onset, very severe, localized to the right lower quadrant with rigidity, distension and tenderness over the area of pain. But there is no fever or leucocytosis, and pelvic examination reveals a tender tumor to the right of the uterus, bulging downwards from the vaginal vault. Thus again the signs of pelvic disease predominate, and attention is called to the fact that the cause of the attack must be there.

*Typhoid Fever.* — Outside this group of localized disturbances in the stomach, intestines, gallbladder, kidney or pelvic organs, producing a clinical picture that may be confused with that of acute appendicitis, there are two general infections, that sometimes lead to error. One of these, typhoid fever, presents its chief manifestations in the intestine; the other, pneumonia, in the lung. It seems incredible that either one should ever be mistaken for acute appendicitis, but now and then this does occur. Typhoid fever does not give rise to abdominal pain, as

a rule, except when perforation complicates, and this is a late accident. Nevertheless pain and tenderness appearing early, greatest in the right lower quadrant, with fever and all the classical signs on physical examination, at times have caused sufficient resemblance to acute appendicitis to lead to unnecessary operation. Then the swollen Peyer's patches found in the lower ileum and the cæcum have made plain the diagnosis of typhoid. But it should be possible to distinguish one from the other without a visible demonstration of the pathological lesion. There is commonly a history in typhoid of disturbed health for days preceding the acute abdominal pain, of malaise, dull headache, loss of appetite, disturbed sleep, weakness. By the time that pain appears, fever is found, and it tends to rise steadily each day. But the pulse is not quickened in proportion to the temperature. Leucocytes are not increased. Diarrhœa is frequently present with stools of pea-soup consistency. Rose spots may be discovered if search is made for them, and the spleen may be found enlarged. With all these peculiar deviations from the usual picture of acute appendicitis, it is justifiable to wait at least a day or two before operation to observe developments and collect further evidence. If time is taken for investigation, a positive Widal reaction may be found, or blood, urine or stool cultures may show the typhoid bacillus before the Widal appears.

*Lobar Pneumonia.* — Lobar pneumonia at the right base sometimes produces pain referred to the right lower abdomen, so much like that of acute appendicitis as to prove extremely deceptive. Even the characteristic muscle spasm in that area may accompany the pain. But there are several important details that ought to save the diagnostician from disaster. First, pneumonia commonly makes its onset with chill followed

by high fever, manifestations most unusual in acute appendicitis. Second, the leucocytosis found early is much higher than that of appendicitis, and usually exceeds 20,000. Third, the signs over the right lower quadrant may be suggestive but are rarely so definite as in acute appendicitis, while the signs over the right pulmonary base, impaired movement, decreased resonance, bronchial breathing, fine crackling râles, ought to prove that this is the real site of the disease. If these signs always were found over the lung at the beginning, probably there would never be any difficulty about the diagnosis. But it is precisely in the case where the patch of consolidation is deep-seated, and the resulting pleurisy is diaphragmatic, that the pain is referred to the abdomen rather than to the chest wall, and the diagnostic physical signs of pneumonia are not found. Sudden violent onset with chill, high fever, high leucocyte count and atypical findings over the right lower quadrant at least should inspire caution and prompt delay about operation until there is time for further observation, and ultimately the usual characteristic signs at the pulmonary base will appear.

### TREATMENT

There is no medical treatment for acute appendicitis. But when the case is first seen, while the diagnosis is still uncertain and appendicitis only suspected, there are a few well-tried and trustworthy rules for the physician's guidance. (1) Keep the patient at rest in bed and under close watch. (2) Note the temperature, pulse and leucocyte count at once and repeat these observations every two hours. (3) Meantime give no food at all and water only in small quantities. (4) Wash out the stomach, if vomiting is persistent, and give an enema, if the patient feels the desire to have the bowel emptied. (5) Place an ice-

bag over the region of pain and keep it there. (6) Give no cathartic drugs of any kind; they increase peristalsis, when rest of the bowel is essential, and spread the inflammation, when every effort should be made to limit it. (7) Give no opiates. They relieve pain, but in doing so they obscure the signs that point out what course the disease is taking. (8) As soon as the diagnosis is definite, as it ordinarily ought to be within twelve hours from onset, have the appendix removed without further delay. Do not wait for complications to appear before advising surgery. Statistics prove beyond question that the earlier the operation, the lower the mortality, and the later the operation, the less likelihood that it will save life.

### CHRONIC APPENDICITIS

The popular conception of chronic appendicitis is of a smouldering fire, burning at all times but only now and then flaring up into the flames of an acute attack. But this is not correct. More often the condition is like that in chronic endocarditis. There is no active inflammation going on constantly in either case, but there are deformities and adhesions resulting from previous acute inflammation that cripple the parts concerned and interfere with the proper performance of their functions, and there are changes in the tissues that make them less resistant to infection so that new invasions succeed more readily and new acute attacks occur with greater frequency. In chronic appendicitis the disturbances of function that result concern the gastrointestinal tract. What these are and how they may be recognized is described in Chapter X of this book entitled "Appendix Dyspepsia"; and to this the reader is referred.

## CHAPTER XXI

### LOCALIZED INFLAMMATION OF THE INTESTINES (CON'T.)

#### DIVERTICULITIS

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A diverticulum is defined as a blind tube or sac branching off from some cavity or canal. Several little pouches or sacs of this sort may project from the wall of the large bowel, and when they are numerous, the condition is call *diverticulosis*. They occur most frequently in older people, past fifty, and may be found along the course of the entire colon but more often along the sigmoid than in any other part. The presence of such diverticula is of no consequence unless they become infected and inflamed. Then the disease known as *diverticulitis* results. Ordinarily diverticulosis itself produces no symptoms and is not to be regarded as a disease.

#### CLINICAL HISTORY

If one or more of these little pouches becomes filled with fæces and unable to expel the contents back into the lumen of the bowel, infection follows and acute inflammation. Then the result is pain in the abdomen, usually felt in the left lower quadrant. With this there is a little fever and disturbance of bowel function. Most often this disturbance is constipation,

but diarrhoea may result instead with tenesmus. As a matter of fact the history shows that constipation frequently has preceded the attack of pain and apparently has been a factor in its production. The abdomen over the left lower quadrant is tender and more resistant than normal. Sometimes an indistinct mass can be palpated. Such an attack may last for a few days and then disappear; the patient may recall several such recurrences with good health between and no symptoms of any kind in the interval.

At any time a recurrence may prove more severe than the ones that have preceded with sharper pain, more definite swelling and tenderness and higher fever. Such an attack may lead to abscess formation so that immediate operation is required, or else the pus may break through into the rectum and discharge by that route, or persistent septic fever may follow with danger of a general peritonitis and a fatal outcome, if rupture occurs into the general peritoneal cavity. Even without abscess formation repeated acute attacks may result in gradual thickening of the bowel wall by chronic inflammation with narrowing and obstruction and obstinate constipation. In such cases a palpable tumor ultimately may be found in the left lower quadrant, tender and resistant, seriously interfering with bowel evacuation and so with general health, thus resembling closely a malignant growth.

### DIAGNOSIS

Diverticulitis occurs as a rule in patients advanced in years. They have long suffered from chronic constipation, and they are likely to describe one or more or a series of attacks preceding, all characterized by pain in the left lower abdomen with soreness, tenderness and fever. Frequently they show on physi-

cal examination a thickening and resistance or even a definite tumor at the site of their pain. During an acute attack increase in the number of leucocytes may be found. X-ray films after a barium enema will show obstruction in the sigmoid region and a definite filling defect, possibly also numerous diverticula in this portion of the bowel. Proctoscopy is of service chiefly by eliminating cancer of the rectum from the diagnostic possibilities, but the real pathological process in diverticulitis usually is too high for direct observation by this instrument.

Only two other diseases offer difficulty in differential diagnosis. *Appendicitis* is at once suggested by the history of recurrent painful attacks with fever and leucocytosis, but the site of the pain and the physical signs in all these attacks in the left lower quadrant instead of the right should prove that the pathological process is not in the appendix, while the demonstration by x-ray films of obstruction and diverticula in the sigmoid colon shows where the cause of the clinical picture really is situated. *Cancer of the sigmoid*, rectosigmoid junction or rectum is not so easily eliminated. Digital exploration of the rectum may reach a cancer in that part of the bowel and be able to identify it, but can not give information about a tumor produced by diverticulitis because it lies too high. The same is true as a rule of the proctoscope. The x-ray films demonstrate not only obstruction of the bowel and a filling defect but also diverticula. The segment of intestine involved by diverticulitis is also longer in most instances than that involved by carcinoma. It is difficult, however, to decide in advance of operation, but this fortunately is indicated after either diagnosis; even at the operating table, with the abdomen open, sometimes it is impossible to say which process is responsible,

and the question is not settled until after histological examination of tissues removed.

### TREATMENT

This is not always the same, because different groups of cases call for a different plan of management. *First*, where the complaint is of a series of attacks of pain localized to the left lower abdominal quadrant, of moderate intensity and brief duration, medical treatment usually is sufficient to give relief. The diet should be soft and bland and with little residue in order to avoid irritation to the lower colon by fragments of food particles that may lodge in the little pockets along the course of the bowel and so induce inflammation. In addition constipation must be prevented by a mild laxative taken regularly, to avoid stasis in the sigmoid and rectum, such as one of the mineral oil preparations with agar. By these simple means comfort usually is assured and recurrence of an attack is prevented.

*Second*, there is a group of cases where increased severity of pain, higher and more persistent fever, greater tenderness and swelling and increased leucocytosis indicate supuration and accumulation of pus in a localized abscess. Then incision and drainage become imperative without too much delay.

*Third*, in still another group with a history of greater or less degree of bowel obstruction, accompanied by soreness, tenderness and aching pain in the left lower abdomen and with a palpable tumor in that region, resection of the thickened, diseased portion of the sigmoid may be required.

The following case histories illustrate these different groups as met in practice:

1. A woman, aged 76, complained that ever since an illness a year previous, characterized by soreness in the left side of her abdomen and fever lasting for several days she had continued to have similar attacks at intervals, but never lasting long, with good health between them. Her bowels were constipated, did not move every day and never freely, and taking laxatives only increased her discomfort. She was frequently disturbed at night by a sense of fulness in her abdomen with belching of gas and its passage by rectum. She always felt worse after her bowels moved freely, better when they did not. During the year's illness she had lost considerably in weight and strength. Physical examination of the abdomen showed normal contour, no distension or rigidity, slightly greater tenderness over the left side than over the right but no palpable mass. X-ray films after a barium enema demonstrated numerous outpocketings along the course of the sigmoid and descending colon, typical multiple diverticula, slight narrowing, well-localized tenderness and moderate fixation of the bowel at the lower end of the descending colon with dilatation just above this level. The diverticula were most numerous in the region of delayed filling and narrowing of the bowel, and one definite pocket was outlined in the area of tenderness and fixation. This patient was given a soft, non-residue diet, much like the second list recommended for gastric ulcer. The only medication advised was a petrolagar preparation each night as a laxative. On this plan her symptoms quickly disappeared. Over two years have passed, and the trouble has never recurred.

2. A man, aged 50, seen in consultation, had been ill for two weeks with indefinite discomfort at the outset but later on with severe pain in the left lower abdomen and fever as high as 102° F. His leucocyte count was 16,000 with 93 per cent. polymorphonuclears. On physical examination decided tenderness and resistance was found in the left lower quadrant over the region of pain. A diagnosis of acute diverticulitis was made, and operation was advised. The abdomen was opened a few days later by Dr. Emmet Rixford. A tumor mass was found at the brim of the pelvis involving the sig-

moid and mesentery, so adherent it could not be drawn up into the wound. No attempt was made at removal, but drains were inserted, and the wound left open. Two days later there was profuse discharge of very foul pus. This continued for a month but gradually ceased. The patient ultimately recovered his health completely and has remained well since.

3. A woman, aged 72, complained chiefly of a sense of soreness in her left lower abdomen with progressive weakness for a year or two before this soreness began to be noticed. Very definite tenderness was found at the site of pain but no palpable mass. This attack disappeared after rest and laxatives and left her comfortable. But a few weeks later she reported a recurrence of her symptoms with cramps, loss of appetite and a little fever. This second attack of pain and soreness in the left side of the abdomen proved more severe than the first, the suffering as severe, she said, as that experienced in childbirth years before. Both attacks were accompanied by diarrhoea for two or three days, though ordinarily she was constipated and required a laxative habitually. Physical examination showed the abdomen distended, with rigidity and greatest tenderness in the left lower quadrant, but no palpable mass. When a barium enema was given for x-ray examination, considerable difficulty was experienced in persuading the material to pass through the sigmoid region, and by the fluoroscope there was noted constantly a filling defect at this point, which could not be overcome by manipulation. On the films this defect measured 6 or 7 cm. in length, and in this region of narrowing many diverticula were seen. At operation a mass was revealed involving the lower portion of the sigmoid, measuring 8 cm. in length  $\times$  5 cm. in diameter, bound down by numerous adhesions. These were freed, and the mass excised, and an end to end anastomosis was done with a Murphy button. On gross inspection the mass was composed to a considerable extent of inflammatory tissue which involved the entire circumference of the large bowel. The report of the pathologist on the specimen removed showed greatly thickened walls and moderate narrowing of the lumen, a number of diverticula, some of which contained small

fæcaliths, extensive lymphocytic and eosinophilic infiltration of the submucosa with fibrous thickening of the latter and much hypertrophied muscularis. The mucous membrane was intact. There was no evidence of malignancy. The anatomical diagnosis was "chronic diverticulitis of the colon". Unfortunately this patient died five days following operation from a pulmonary complication.

## CHAPTER XXII

### ULCER AND DIVERTICULUM OF THE DUODENUM

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Ulcer occurs more frequently below than above the pylorus, and in this situation it becomes anatomically a disease of the intestine. Nevertheless clinically it resembles gastric ulcer so closely that it is difficult to distinguish one from the other by history and effects upon digestive functions. In fact the two may be found coincident, one in the stomach and one in the duodenum, or the ulcer may lie across the pylorus, partly inside and partly outside the stomach. In the duodenum, when the ulcer is there and nowhere else, it is situated almost invariably in the first portion, known as the bulb, within the first inch of the intestine's course. Furthermore its common site is the anterior or posterior wall, not the right or left wall, which form the continuation of the greater and lesser curvatures of the stomach. Like the gastric ulcer, the duodenal is

rounded or slightly oval. The size varies, but the very large ulcers sometimes seen in the stomach are much more rare in the duodenum. Ulcer on the posterior wall has the head of the pancreas behind it and in case of deep penetration becomes adherent to this organ, which forms its floor and prevents perforation. Ulcer on the anterior wall has no such direct protection and more frequently perforates into the peritoneal cavity, though this catastrophe may be prevented by adhesions to the gallbladder, the liver or the colon.

### CLINICAL HISTORY

The same characteristics are observed in the story of ulcer below the pylorus as in that of ulcer above, namely, chronicity, periodicity, rhythmic sequence of events and peculiarity in the nature of the symptoms. In fact this story is even more typical of duodenal than of gastric ulcer. The account of stomach trouble goes back for months or years with remissions or intermissions and then again exacerbations, the chief complaint being pain coming on several hours after a meal or in the middle of the night, relieved by the next meal or by a glass of milk or by bicarbonate of soda, the symptoms besides pain being a sense of burning, belching of gas, sour eructations, nausea and sometimes vomiting of very sour, irritating fluid which always gives relief. Details of these various features are given in the account of gastric ulcer and need not be repeated here, because they are so much alike, regardless of the ulcer's exact site.

### GASTRIC ANALYSIS

Some of the highest secretory values are found after a test meal in duodenal ulcer. Even in the fasting contents the total

amount of secretion is unusually large, and the proportion of free HCl also is much increased. After a fractional test meal the acidity also runs above the average normal, but these findings by themselves are never diagnostic and are of importance only when considered with all others. Furthermore it must not be assumed that the amount of HCl is always high. This is the rule, but normal or even subnormal curves may be found, exceptionally even achlorhydria.

### STOOL EXAMINATIONS

Occult blood in the stools, as explained in the chapter on gastric ulcer, is not evidence of the presence of an ulcer but simply of its activity. There may be none found even when duodenal ulcer is present, because they do not always bleed. On the other hand, when slight bleeding is persistent and goes on steadily as shown by a reaction for occult blood day after day, ulcer is the most probable explanation, but even so this does not tell on which side of the pylorus the lesion lies. There are other causes, too, for this laboratory finding, and it may be due to bleeding anywhere in the digestive tract from teeth to anus, or to nasopharyngeal disease or even to rare meat taken in the diet with no bleeding anywhere.

### X-RAY EXAMINATION

Before this method of investigation was developed to its present state of accuracy, there was no way to diagnose duodenal ulcer with any degree of certainty, for no other test is conclusive. Now it is possible by fluoroscopy and x-ray films to recognize its presence with almost absolute correctness. The duodenal bulb is so small as compared with the stomach that the deformity produced by an ulcer usually is detected

with ease. There is a "niche" or defect noted in the contour of the bulb, or in an old chronic ulcer repeated scarring or muscle spasm produces a deformity that makes the bulb resemble a clover leaf.

Valuable and indispensable as x-ray examination is in gastric ulcer, it is even more important and conclusive in duodenal. Even so, errors sometimes occur, and the method is not absolutely infallible.

### DIAGNOSIS

By the clinical history, by the negative physical examination and by gastric analysis it is usually possible to infer that the patient has a peptic ulcer, but only by x-ray examination and discovery of a deformed duodenal cap can it be proved that an ulcer exists, and that it is duodenal rather than gastric. When clinical history, negative physical examination and gastric hypersecretion all point to ulcer, but the x-ray reveals none, then it becomes probable that some extragastric disease is responsible for the patient's symptoms, such as chronic inflammation of the gallbladder or the appendix, or that a disordered nervous system has produced them by disturbing gastric secretion, without any discoverable organic disease anywhere.

How to decide what this extragastric cause really is, or whether one exists at all, has been considered in detail in the chapters on gastric ulcer and on reflex dyspepsias and on gastric neuroses, and to these the reader is referred. It should be kept in mind, however, that a diseased gallbladder is at least as common a cause of pain in the upper abdomen with persistent indigestion as duodenal ulcer is and possibly an even more common one, in spite of the evil reputation the latter has acquired.

## TREATMENT

The location of an ulcer below the pylorus in the duodenum rather than above it in the stomach does not alter in any way the general plan of treatment recommended as most efficacious. Whether this is carried out with the patient up and about or at rest in bed, diet, alkaline powders, olive oil and belladonna, as advised for gastric ulcer, constitute the measures ordinarily indicated. The severity of the patient's suffering, the response to treatment and the previous duration of symptoms all influence the decision as to which method is best for the individual case. There is no plan suitable for all, regardless of circumstances.

## COMPLICATIONS OF DUODENAL ULCER

These are not as numerous as the ones observed in gastric ulcer, but those that occur are just as serious and dangerous. Three such complications menace the patient, when the ulcer is below the pylorus, as frequently as when it is above, namely, pyloric obstruction, hæmorrhage and perforation. But the danger of malignant degeneration is practically nonexistent, for cancer developing on the base of a duodenal ulcer is extremely rare.

*Obstruction of the Pylorus*

Obstruction of the pylorus by an ulcer lying below it presents no features that distinguish it from all other causes. The symptoms of obstruction are the same regardless of which side of the pyloric ring the lesion originates that produces them. Gradual interference with the passage of food leads to more constant pain, no longer rhythmic in its repetition, to vomiting in the effort to rid the stomach of its accumulating

contents and to gradual loss of weight and strength by progressive starvation. By physical examination the diagnostic signs are a visible peristaltic wave across the stomach from left to right and dilated contour of the organ, frequently observed through the abdominal wall without any device to produce them, but more certainly after filling the stomach with carbonic acid gas. The stomach tube used for gastric analysis finds a quantity of refuse that must be removed by lavage before any reliable determination of gastric secretion can be made. After such cleansing a test meal usually gives the findings characteristic of chronic gastritis, induced by the food retention. X-ray films demonstrate conclusively the obstruction at the pylorus but do not prove that a duodenal ulcer is responsible, for the barium can not pass far enough to outline successfully the contour of the duodenal bulb. Unless previous history and diagnostic investigation have made clear the presence of a duodenal ulcer, the exact cause of the obstruction must remain temporarily undetermined. As emphasized in the consideration of gastric ulcer, the discovery of pyloric obstruction does not mean immediate resort to surgery is imperative, for inflammation, œdema and spasm are as important factors in its production as ulcer scar tissue and deformity, and these frequently can be overcome by rest, lavage, Sippy diet and alkaline medication, so that the pylorus again becomes competent in spite of the duodenal ulcer. Gastroenterostomy ultimately may become necessary but should never be the first resort before other measures have been tried.

### *Hæmorrhage*

Blood discharged from a duodenal ulcer finds its way out of the body through the bowel more often than through the

stomach, by mælena rather than by hæmatemesis. The amount may be small but persistent, discovered only by chemical tests of the stool for occult blood, or it may be large enough to be seen on gross inspection of the bowel discharges as black lumps mingled with the other excrement, or it may be so large that the entire stool consists of a black, sticky mass, resembling tar. But blood found in the stools does not necessarily come from a lesion in the intestine. It may leak first into the stomach and then pass out through the pylorus into the bowel without hæmatemesis, if the amount is small, or following hæmatemesis, if the amount is large. Blood discharged directly into the duodenum from an ulcer may never run back into the stomach at all, and so no vomiting of it occurs, but all escapes through the intestine. Mælena without hæmatemesis thus suggests a duodenal origin rather than a gastric, but this conclusion is not altogether reliable, because hæmatemesis may take care of most of the bleeding, even when the source is a duodenal ulcer below the pylorus, or in exceptional instances all the blood thus lost may escape by the stomach. Thus when mælena occurs alone or greatly exceeds hæmatemesis, it is probable but not certain that it means duodenal ulcer. A history of chronic indigestion preceding a large hæmorrhage from the bowel usually gives a clue as to its cause, but a duodenal ulcer may be latent and cause so few symptoms that the hæmorrhage comes first without warning. Such an incident speaks for ulcer rather than for any other lesion, but additional evidence is required for certainty. The fact that the stools are black, sticky and tar-like, indicating that the blood has traveled far from the point where it was first discharged, is not diagnostic of either gastric or duodenal ulcer, because bleeding into the stomach may result from rupture of dilated

veins in the lower œsophagus, produced by cirrhosis of the liver or splenic anæmia, and the blood thus poured out may be mainly discharged by the bowel. Blood escaping from lesions lower down in the intestine, as in typhoid fever, ulcerative colitis, cancer of the colon or intestinal polyps, does not travel so far to reach the rectum and, therefore, does not undergo as a rule the chemical changes that make its appearance black and tar-like. Dark clots are commonly observed, but there is red blood also mingled with the stools. Statistics prove that hæmorrhage from a duodenal ulcer is not often fatal, and that the mortality from this complication does not exceed 2.5 per cent. Treatment should, therefore, be medical by choice and carried out on the same plan as that advised for hæmorrhage from gastric ulcer. At the outset it is often impossible to distinguish the exact source of the bleeding, whether above or below the pylorus, and the immediate indication is to check the hæmorrhage no matter where it originates.

### *Perforation*

This is a more common accident in duodenal than in gastric ulcer. It occurs in nearly all cases through the anterior wall. The symptoms and signs it produces are in no way different from those in gastric ulcer, and the same indication exists for treatment by surgery as promptly as possible after diagnosis. Usually it is impossible to state in advance whether the stomach or the duodenum is the site of the perforation, unless previous investigations, particularly x-ray films, have proved whether the ulcer was gastric or duodenal, but it makes no difference in the procedure required for saving the patient's life. Perforation of a duodenal ulcer, like hæmorrhage from it, may be the first event of sufficient importance to attract attention, because the

preceding course has been so insidious. Delay about operation, therefore, should never result from the absence of previous history.

### DIVERTICULUM OF THE DUODENUM

This is not an uncommon finding in x-ray films, but the incidence varies according to the statistics of different observers all the way from five-tenths of one per cent. to five per cent. of all cases where x-ray examination of the gastrointestinal tract have been made. Probably the incidence actually does not exceed two per cent. The diverticulum varies in size in different instances, from 0.5 to 3 cm. in length. Usually it is single.

There has been considerable controversy as to whether such a diverticulum is congenital or acquired, but the fact that it is found more frequently as age advances speaks for its origin from disease or degeneration rather than from inherited developmental defect. Yet it is possible that inherited weakness permits the formation of the diverticulum in one individual and not in another. Like diverticula of the sigmoid most cases are seen in patients past 50, and the greatest frequency is at 70 or later.

The lesion that most frequently precedes the appearance of a diverticulum is a duodenal ulcer, and the two are associated in many instances. Nevertheless diverticulum may be found when no other abnormality of this part can be demonstrated. As regards the symptoms it produces, there are none that are peculiar to it, and most investigators agree that the manifestations observed resemble those of duodenal ulcer and can not be distinguished from them. Others, however, question this statement and think that the condition can be recognized

even previous to its demonstration by x-ray films. But this would appear to be true only when the diverticulum has been distended by bowel contents or has become inflamed. Finally the opinion has been expressed that no symptom whatever can rightly be attributed to the diverticulum itself, and that when symptoms arise they are always the result of diverticulitis or an associated duodenal ulcer or pylorospasm secondary to some other pathological process.

The practical question is always "What shall be done when x-ray films show a duodenal diverticulum?" The weight of opinion favors letting it alone. If medical treatment for the associated ulcer or for pylorospasm does not relieve symptoms, then the only other recourse is surgical removal of the diverticulum. But this should never be recommended hastily, or until all other measures for relief have been tried.

## CHAPTER XXIII

### CANCER OF THE INTESTINES

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Cancer of the intestine is less common than cancer of the stomach, but in any case of chronic digestive disturbance the symptoms may mean cancer, if not above the pylorus then below it, in the long passage between that gateway and the anus. Most often these symptoms are produced by interference with bowel functions, when the intestine is the site of the neoplasm, but they may suggest gastric more than intestinal disease, particularly when the growth is in the small bowel, but at times even when it is in the large bowel. Because there are a number of different situations at which cancer of the intestine develops, and because its location has much to do with the character of

the manifestations, subjective and objective, upon which its recognition depends, each requires separate consideration. There is no one description that suffices for all.

### CANCER OF THE DUODENUM

This is a rare site for malignant disease. Nevertheless the symptoms produced when cancer does develop here are so striking that their significance usually is easy to interpret. Like cancer anywhere in the body, the growth in the duodenum causes little by little such general manifestations as anæmia, cachexia, loss of weight and strength, but there are two other evidences that serve to call attention to its situation, pain and a palpable tumor. Pain and tumor usually are located in the right upper abdominal quadrant, more accurately in the right hypochondrium and the epigastrium. But certain special symptoms are produced, according to the part of the duodenum involved, and these help to determine its exact site. Boas first classified duodenal growths with reference to the biliary papilla, into three varieties, supra-papillary, circum-papillary and infra-papillary. Each has its own characteristics.

#### *Supra-papillary Cancer of the Duodenum*

In supra-papillary cancer of the duodenum no localizing symptoms become prominent until stenosis of the bowel results. Then the manifestations of its presence become very similar to those of pyloric stenosis with dilated stomach, and usually it is impossible by symptoms and signs alone to decide whether the cancer originates at the pylorus or below it. In such a case with the general evidence of cancer such as furnished by loss of color, weight and strength, with localizing symptoms such as pain and a palpable tumor in the right

hypochondrium, with special symptoms such as disturbance of appetite, fulness and distress after eating, nausea and ultimately repeated vomiting of retained food, with the physical signs of dilated stomach such as its visible outline, a peristaltic wave across it and a succussion splash over it, the diagnosis of a neoplasm obstructing the pyloric orifice becomes fairly certain. Use of the stomach tube for gastric analysis reveals food retention and decreased secretion, but does not give any evidence to prove that the obstruction is below and not at the pylorus. Even x-ray films, while they confirm the fact that the stomach is dilated and empties slowly, if at all, do not prove the site of the obstruction unless they succeed in showing also a pouch of dilated duodenum beyond the pyloric orifice. In any event surgery is indicated, and no error can be made by advising it. In attempting to reach a conclusion in advance of operation, a point of value in differentiation is the much greater frequency of cancer of the pyloric end of the stomach than of the duodenum.

A woman, aged 66, always previously well had been complaining for two months that food caused pain soon after she ate it. She vomited occasionally, and this gave relief, and vomiting had occurred more and more frequently as time went by. She had lost much weight, and her bowels had become so constipated they would scarcely respond to physic. Her abdomen was found distended and tympanitic, but there was no tumor visible or palpable except the outline of the dilated stomach which could be distinctly observed as a large bag with the greater curvature halfway between the navel and the pubes, with a constant succussion splash over it and a partial peristaltic wave across it from time to time. Gastric analysis after lavage showed total acidity 28 and no free HCl. X-ray report stated that at six hours and as late as twenty-four hours the stomach retained practically all the barium meal, and the pyloric end of the

stomach could not be clearly outlined because of marked fluid retention. A diagnosis was made of carcinoma at the pylorus and operation advised. This revealed no growth in the stomach but one beyond it in the duodenum, supra-papillary, with great dilatation of the duodenum above it. Metastases prevented the growth's removal, but a gastroenterostomy was done. The patient lived only a month afterward.

### *Circum-papillary Cancer of the Duodenum*

Circum-papillary cancer of the duodenum furnishes a new symptom that can not be overlooked, jaundice. This is often insidious in onset, making its appearance before pain or digestive disturbances have occurred or at least have become prominent. But it is progressive and ultimately profound, and then the predominating symptoms become those of obstruction of the common duct rather than those of pyloric or duodenal obstruction. The diagnostic problem thus presented is one of the most difficult in clinical medicine. Differentiation must be made from the other common causes of obstructive jaundice, such as cancer originating in the common duct rather than in the duodenal wall, or in the gallbladder, the head of the pancreas, the pyloric end of the stomach or the liver itself as well as a gall-stone impacted in the common duct. There is absolutely no trustworthy evidence to identify the cause of the jaundice as a malignant growth in the duodenum involving the biliary papilla. The sifting out of one possibility from the others requires the most careful consideration of the patient's history, physical examination, gastric contents, stools, blood count, blood bilirubin, urinalysis, as well as x-ray examination of the gastrointestinal tract. In spite of the most thorough investigation the distinguishing marks of circum-papillary duodenal cancer are so uncertain that operation often becomes

the last resort for diagnosis as well as for treatment. The rarity of this form of disease, however, as compared with some of the other causes of obstructive jaundice, should be kept in mind in estimating the possibilities.

*Infra-papillary Cancer of the Duodenum*

Infra-papillary cancer of the duodenum introduces still other characteristic symptoms and signs into the clinical picture, because obstruction of the duodenum below the papilla leads to interference with the normal downward course into the intestine of the bile and pancreatic juice, which then are regurgitated into the stomach and rejected by vomiting. This copious vomiting of dark brown material resembling bile, neutral or alkaline in reaction and responding to chemical tests for bile, thus becomes a diagnostic feature. This same material will be found in the fasting stomach contents in the morning, even though lavage has been done the night before. The stools usually are light colored or even white because bile is deficient or absent in the intestinal contents. On physical examination bulging is noted sooner or later in the epigastrium, due to dilatation not only of the stomach but also of the duodenum above the point of obstruction. The difficulty about visualizing accurately the course of the duodenum in x-ray films makes this method of investigation of doubtful value in recognizing this disease, but it may show dilatation not only of the stomach but of the duodenum beyond the pylorus without demonstrating just where the cause is situated. The persistence of vomiting and the peculiar character of the vomitus, the ultimate development of a palpable tumor in the epigastrium, the absence of bile from the stools, the demonstration by fluoroscopy and films of a dilated stomach and upper duodenum

even though the site of the obstruction can not be shown, together with the gradually increasing cachexia and malnutrition of the patient, are the evidence depended upon to identify infra-papillary cancer of the duodenum. Such evidence at least justifies advising surgical exploration, for no other plan of treatment affords any hope.

#### CANCER OF THE JEJUNUM AND ILEUM

According to all observers cancer is exceedingly rare in these parts of the small intestine, which seems singularly exempt below the duodenum from attack by malignant neoplasm, as compared with the stomach above and the large bowel below. It is fortunate that this is so, because the entire extent of the small bowel offers great difficulties to investigation by x-ray or any other method, and diseases of this portion of the digestive tract are more or less hidden from observation. It is a source of satisfaction, therefore, to realize that cancer of the jejunum or ileum is almost never observed, not only by the clinician but also by the surgeon or the pathologist, and in the investigation of any case suspected to be intestinal cancer from the outset the probabilities are all against a growth above the ileocecal valve.

#### CANCER OF THE COLON

Beyond the ileocecal valve the incidence of cancer rapidly increases. But there are sites in the course of the large bowel that seem especially liable to attack by malignant disease, though it may develop at any point between these. These areas are the cæcum, the hepatic and splenic flexures, the sigmoid and the rectum. While there are symptoms common to all cancers of the colon, there are also peculiarities about the

evidence presented in each locality that aid in determining its situation and that, therefore, make it profitable to consider each by itself.

### *Cancer of the Cæcum*

*Clinical History.* — With cancer originating in the cæcum, the early symptoms frequently are those of indigestion due to increasing interference with the progress of feces and gas. Reflexly gastric functions are disturbed with loss of appetite, heaviness and discomfort after meals, epigastric distress, belching of gas and a feeling that the food remains too long in the stomach. Gradually the disturbance affects intestinal functions as well. Then the complaints most often heard are of increasing constipation, flatulent distension and recurring attacks of colic. These are common to cancer in any part of the colon, not limited to cancer of the cæcum, but they vary in different cases in the degree to which they become annoying and significant.

(a) *Increasing constipation* and difficulty about securing a daily evacuation even by laxatives is not as prominent in the history of cancer of the cæcum as when the growth is at some other site. The reasons for this are the normally wide lumen of this part of the large bowel, which allows the contents to pass even in spite of the gradual development of obstruction by a neoplasm and the fact that a growth in this situation involves as a rule only a portion of the wall and does not completely surround it. Complete occlusion is thus a late development at this point, if it occurs at all, and even obstinate constipation less outstanding a symptom than when cancer involves the descending colon or sigmoid.

(b) *Flatulent distension* is so common that by itself it has

little significance. It usually is caused by the retention and decomposition of fæcal masses in the large bowel, but delay in evacuation, which permits this to occur, does not necessarily mean neoplasm of the cæcum or of any other area in the colon. If it persists, however, or continues to recur after castor oil or some other brisk purgative has caused it temporarily to disappear, it has a significance that can not be overlooked. This may be the only symptom of any prominence until late in the course of the disease.

(c) *Recurring attacks of colic* are likewise not so common when cancer involves the cæcum as when it develops at some point further along in the colon. Such attacks may never occur at all, but when they do, usually it is in association with alternating constipation and diarrhœa. Following a period of difficulty about securing an evacuation even in response to laxatives, sharp and colicky pains appear, followed by frequent bowel discharges for a day or two. Then follows another period of constipation, terminating once more, sooner or later, in a recurrence of colic and diarrhœa. The absence of such attacks from the history does not exclude the possibility of cancer of the cæcum, but their presence is always of serious import.

(d) *Anæmia*. — In addition to the foregoing complaints that are common to cancer in any part of the colon, there is another distinctive feature about cancer of the cæcum, less frequently observed when the neoplasm is further along. This is advanced secondary *anæmia*, and it may attract attention before any disturbances of intestinal function have arisen that suggest a malignant growth in the bowel. The explanation offered for this is the large size which growths in the cæcum attain and their tendency to ulcerate. From an extensive

broken down area thus resulting blood oozes more or less constantly, and through it micro-organisms enter. Thus bleeding and infection combine to produce anæmia, and the symptoms of this are frequently out of proportion to those implicating the bowel. Such anæmia, however, does not always result from cancer of the cæcum, and this manifestation is not essential to the diagnosis.

*Physical Examination.* — Cancer of the cæcum usually produces a growth of considerable size, and the discovery of this by the patient may precede any symptoms serious enough to cause alarm. A palpable tumor is nearly always found by the physician by the time advice is sought, regardless of whether the patient is or is not aware of its presence. It lies usually in the right lower quadrant below the level of the navel, sometimes as low as the inguinal region, but it may be found higher. The typical tumor is hard, irregular or nodular, frequently tender, usually freely movable but at times fixed by adhesions. But it possesses no features that positively identify its character or location without supplementary evidence of other kinds. The one other common finding on physical examination is tympanitic distension of the abdomen, and this may be so extreme that it obscures the tumor until after gas has been removed from the bowel by laxative or enema or both.

*Laboratory Examinations.* — From these not much information of direct value is obtained, but neglect to add their evidence to that obtained in other ways may lead to disastrous error in diagnosis. (a) Gastric analysis frequently shows decreased or absent secretion of free HCl, even when the cancer is as far away as the cæcum, but not always, and secretion may be found within normal limits. (b) Stools show blood, visible or occult, persistently, if the neoplasm has ulcerated and

presents a raw surface to the lumen of the bowel, but hæmorrhage of any consequence from cancer of the cæcum is an unusual complication. Blood in the stools, either bright red or in dark clots or even when detected only by chemical tests, if persistent, always is a suspicious sign and calls for careful investigation as to its meaning. (*c*) Urine analysis is of value in distinguishing a tumor of the cæcum found high in the abdomen from a tumor of the right kidney that has prolapsed that organ, for the site at which a tumor is palpable never identifies its origin. (*d*) Blood examination is particularly important when cancer of the cæcum is suspected, because of the frequency with which a growth at this situation leads to a severe secondary anæmia. But the blood count may be practically normal even when the tumor is large, because there has been no ulceration, no bleeding and no infection.

*X-ray Examination.* — Whenever cancer of any part of the colon is suspected, this examination should be made after a barium enema, never after a barium meal. When the cæcum is the site of a neoplasm, x-ray films show a definite irregularity of this part and a filling defect there. The barium is likely to be retained longer than normal. If a tumor has previously been palpated, the fluoroscope will prove whether its location is in the cæcum. Thus it is usually made clear by x-ray examination that the cæcum is the seat of some form of pathological lesion, but the character of this may require other evidence for its determination.

*Diagnosis.* — Symptoms of indigestion, gastric as well as intestinal, flatulent distension of the abdomen, recurring attacks of colic and diarrhœa are more characteristic of neoplasm involving the cæcum or the right half of the colon than of that involving the left, while symptoms of obstruction are not so

common and are always later in appearing. Progressive anæmia with or without the preceding symptoms to suggest intestinal disease as the cause, a tumor in the right lower quadrant, usually easily palpable, and demonstration of irregularity and a filling defect in the cæcum are the items of evidence upon which the diagnosis of cancer of this part of the bowel depends. But there are two other diseases of the cæcum resembling this closely enough to cause difficulty in differentiation, chronic appendicitis and ileocæcal tuberculosis. While the clinical history may be similar in all three, *chronic appendicitis* does not produce as large or as distinctly palpable a tumor as neoplasm, not so hard or irregular, but more indefinite and less easily distinguished from the surrounding tissues. The tumor produced by *ileocæcal tuberculosis* may more closely resemble neoplasm, but there is a history, or there are physical signs in most cases of previous disease in the lungs, or x-ray films of the chest show active or healed lesions there. Furthermore x-ray films do not show as definitely a filling defect in the cæcum because of the latter's irritability, which compels the barium to hurry on, while in either neoplasm or chronic appendicitis the barium is delayed even beyond the normal emptying time, and the defect produced by disease is unquestionable.

A woman, aged 40, made particular complaint about her stomach, annoying for a long time previous but worse recently. Shortly after eating, pain began, she bloated terribly, gas was evacuated both by the mouth and the rectum, and then she was much relieved until she ate again. Almost any food she took produced gas and great distress. For this reason she had recently confined herself to liquid diet, which caused less pain but made her lose weight and strength. She had no nausea or vomiting and "a wonderful appetite", but she was afraid to eat solid food. There was no complaint of constipation, and her bowels moved regularly each day

without laxative. Her only other symptom was pain at times in the right side of her abdomen, never severe. Physical examination showed no evidence of organic disease anywhere except in her abdomen, but this was distended in its lower half to the size of a six months pregnancy, pouched forward, tympanitic throughout, showing no signs of fluid. In addition a hard irregular mass was palpable low down in the right inguinal region, the size and shape of a small banana. Examination by vagina was negative. The urine was normal. The blood count also was surprisingly normal with 88 per cent. hemoglobin, 4,520,000 red cells, 12,850 white cells, polymorphonuclears 80 per cent. X-ray films after a barium enema showed the cæcum definitely irregular with a large organic defect. At operation a large carcinoma of the head of the cæcum was found, and the terminal ileum, cæcum and part of the ascending colon were removed. Pathological examination of the specimen showed it was a colloid type of neoplasm with abscesses in the wall of the cæcum and the adjacent fat. Regional lymph nodes showed no evidence of metastases. The patient made a prompt post-operative recovery and remains well one year later.

### *Cancer of the Hepatic Flexure*

At the other end of the ascending colon, neoplasm does not always originate at exactly the same site in every case. It may begin below the flexure in the ascending colon, in the flexure itself, or just beyond it in the beginning of the transverse colon. But as all growths in this region give approximately the same symptoms and signs, there is no advantage in attempting to recognize their exact point of origin. What is important, however, is to determine that the symptoms and signs and particularly a palpable tumor are due to carcinoma of this part of the bowel and not to disease of some other organ in the vicinity. This requires careful investigation, and a great deal depends upon what x-ray films disclose after a barium enema. The

following case history illustrates how easily error in diagnosis was made before x-ray investigation became available.

A number of years ago a woman, aged 45, seen in consultation, complained of dragging pain in the right side of the abdomen and back. She had lost slightly in weight, was constipated and had constant disturbance of digestion. In the right upper abdomen a tumor was found, corresponding in size, shape and location to a prolapsed kidney. She was examined during her illness by seven different physicians and surgeons, who all agreed that the tumor was a prolapsed kidney, the only question being whether it was likewise diseased. Finally it was decided that the organ was tuberculous, mainly on the testimony of a laboratory expert, who claimed to have found tubercle bacilli in the urine. At operation to remove this kidney, it was revealed that the tumor was a carcinoma of the ascending colon originating just below the hepatic flexure. The kidney was in normal position and perfectly sound on gross inspection. The neoplasm was successfully removed, but the patient died a few months later from recurrence.

*Diagnosis.* — This history emphasizes the necessity for painstaking employment of every possible method of investigation in order to determine the character of a palpable tumor in the right upper abdomen. When this resembles a *kidney*, as in the case described, there are two ways to obtain information that will either identify this organ or remove it from suspicion. The first of these is direct examination of the kidney by catheterization of the ureters and by chemical and microscopical tests of the urine obtained from each side and also by x-ray films made after intravenous injection of a dye to visualize the urinary tract. The second way is by x-ray films of the colon after a barium enema. By these methods usually it is possible to determine with certainty whether the tumor is renal or colonic in origin.

A second possible explanation for a palpable tumor found in the right upper abdominal quadrant, resembling cancer of the hepatic flexure, is *cancer of the stomach* at the pyloric end. But this rarely reaches a size where it is not only palpable but large enough to cause confusion with a tumor of the bowel, unless there has been a long preceding history of more or less constant disturbance of gastric functions. By the time it has reached such prominence in the physical examination, pyloric obstruction and dilatation of the stomach have frequently been added to the clinical picture with their characteristic symptoms of pain, vomiting and food retention. Gastric analysis reveals food refuse in the fasting stomach and a deficient or absent secretion of free HCl, and x-ray films of the stomach prove beyond question where the palpable tumor originates.

Another palpable tumor in the right upper quadrant that may suggest cancer of the hepatic flexure sometimes really involves the *gallbladder* instead. Two different pathological conditions of this organ may produce such a tumor. First, it may be due to an accumulation within it of mucous secretion due to blocking of the cystic duct, in which case the palpable mass will be smooth and elastic, or second it may be a carcinoma of the gallbladder itself, and then the tumor will be hard and irregular. Recognition of the site of the palpable mass depends most upon x-ray films of the gallbladder after visualization by the usual dye, if it can find its way into the organ. But much depends also upon previous history, what is shown by the icterus index and van den Bergh test and possibly the presence of definite jaundice.

When no tumor is palpable, a carcinoma in the region of the hepatic flexure, annular in character, may nevertheless proceed gradually to a point, where it causes a fatal outcome

by obstruction, before its presence is recognized. The following case illustrating this likewise was observed in the days before the diagnostic value of x-ray examination in cancer of the colon had been made known. Even to-day this case would probably escape diagnosis without the aid of x-ray films of the colon.

A man, aged 62, complained that he waked up every morning early with a feeling of distention and tightness in his abdomen. This annoyed him so much, even though there was no pain, that he always had to rise and stir about. As soon as his bowels moved, and gas was passed freely, he had no further trouble during the day. This discomfort had been going on for months. Otherwise he felt well and had no complaint to make. There had been no loss of weight. He was a large, rather corpulent man, florid and apparently in perfect health. Abdominal examination, repeatedly made, always failed to show a palpable tumor or even tenderness at any point. The bowels moved regularly every day and various mild laxatives which were advised did not prevent the flatulent distension from occurring each morning. Various changes of diet likewise were of no avail. Finally after taking a dose of castor oil one evening, violent pains were set up and continued. Within a few hours the abdomen became greatly distended, rigid and tender, the pulse rapidly grew irregular and weak, and the patient died. Autopsy showed a rent in the ascending colon near the hepatic flexure with the escape of a large quantity of bowel contents into the peritoneal cavity. A nodular carcinomatous mass was found just beyond the tear, annular, hard, constricting the bowel and practically occluding its lumen.

### *Cancer of the Transverse Colon*

This part of the large bowel seems less liable to malignant disease than the flexures at either end of it, but neoplasm may occur there, and the possibility is always present. Cancer in this location frequently is annular and so itself produces no palpable tumor. When one is found in the upper abdomen,

suspected to originate from the transverse colon, it is sometimes retained fæces that make up the mass recognized and not the growth itself. A dose of castor oil may then remove the tumor. The patient's complaints do not identify the site of the cancer and usually are vague and indefinite, usually about gas and indigestion. Whether a palpable tumor is found or not, the only certain proof that it involves the transverse colon is that supplied by x-ray films. No matter what the history suggests, in no other way can error be avoided. If no filling defect in this part of the bowel is shown by a barium enema, and if there are no symptoms of bowel obstruction in spite of a palpable tumor, a barium meal should supplement the enema, for cancer of the stomach may present a mass palpable anywhere in the upper abdomen, no matter whether it originates at the pyloric end or on the greater or lesser curvature. Even gastric functions may not have been disturbed materially, when the growth originates in the mid-portion of the stomach, not obstructing either orifice, and a tumor easily discovered may appear on physical examination to form a part of the colon, when it is really a part of the stomach.

## CHAPTER XXIV

### CANCER OF THE INTESTINES (CON'T.)

#### CANCER OF COLON (CON'T.) AND OF RECTUM

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#### *Cancer of the Splenic Flexure*

As the transverse colon is left behind, the lumen of the intestine gradually becomes narrower and its carrying capacity decreased. Furthermore the bowel contents become less fluid and more inspissated, more formed and less pliable, the farther they move on toward the rectum, so that they are arrested by a lesser obstacle than in the right half of the large bowel. In the splenic flexure, beyond it in the descending colon, and even more certainly in the sigmoid, the symptoms of cancer thus become those of obstruction, and interference with bowel evacuation is more prominent in the clinical history than indigestion or anæmia or loss of weight. This, however, is not always the case, and there may be no constipation, and on the contrary diarrhœa may predominate in the story. But whether the previous history is of normal bowel movements or constipation or diarrhœa, complete obstruction of the bowel may appear either suddenly or after several attacks of partial obstruction of short duration. On the other hand, it may never appear at

all. Whatever the history, examination of the abdomen may be quite negative except for distension by gas, or sometimes peristalsis may be seen in a coil of intestine that erects itself forcibly in order to pass on its contents. When a tumor is found in the left hypochondrium, the problem becomes one of deciding as to whether the splenic flexure of the colon or the stomach is its site. Then gastric analysis, stool examination, x-ray after barium enema and possibly barium meal are all required in order to reach a conclusion. The methods of investigation to make an accurate diagnosis and the difficulties in the way are illustrated by the following history. It also proves at how early an age cancer of the colon may occur.

A woman, aged 37, had been ailing for about four years, but at the outset with sour stomach requiring the use of soda constantly for relief. For a while she had intervals when the trouble disappeared, but for a year past it had been constant, characterized by heart-burn about two hours after eating, belching, water-brash and pain described as "a dull misery", but so severe recently as to prevent sleep at night. There was no nausea or vomiting and never had been. She had lost weight steadily, from 124 lbs. down to 90 lbs. For nearly a year she had also attacks of cramps in the bowels with diarrhoea. She had never been constipated, even between these attacks.

On physical examination, the patient was found greatly emaciated with abdomen distended. There was a palpable tumor in the left hypochondrium, descending on deep inspiration from beneath the left costal margin nearly to the navel, very hard and slightly irregular. No peristaltic wave was observed over the stomach, even after inflation with CO<sub>2</sub>, but across the abdomen from right to left, in the course apparently of the transverse colon, a peristaltic wave was visible at times, and the patient said she frequently could see, coincident with a cramp, a bulging in her right side, progressing slowly across her abdomen from right to left, forming a large "knot"

in her left side just below the ribs, until something seemed to give way with a rumbling, gurgling noise and then the pain ceased.

When the stomach tube was passed to obtain fasting contents, a few ounces of blood-stained fluid were obtained, and one hour after an Ewald test meal, the bread was found very poorly triturated and in chunks, and the fluid contents were tinged red. There was much thick mucus, a total acidity of 10 but no reaction for free HCl.

X-ray examination showed a small stomach but no definite filling defect. There was great delay in the passing of the meal through the colon at the site of the palpable tumor. At operation, one week after the patient was first seen, a large carcinomatous mass was revealed, involving the splenic flexure with approximately four inches of the transverse colon on one side and two inches of the descending colon on the other. This mass had invaded also the greater curvature and posterior surface of the stomach, which were firmly adhered to the colonic mass. There were numerous metastases like small beads scattered generally over the intestines and mesentery. No attempt was made at removal.

### *Cancer of the Descending Colon*

Beyond the splenic flexure and its immediate vicinity, in the descending colon, cancer gives much the same clinical history as that already described, except that in this part of the bowel obstruction becomes more common, and acute obstruction is often the first event of consequence. If a tumor is palpable, less difficulty is experienced in identifying it, because it lies well below the stomach, and there is no other organ in the vicinity that commonly forms the origin of a malignant growth. On the other hand cancer of the descending colon frequently presents no palpable tumor, and its existence and site are demonstrated only by x-ray films after a barium enema, when repeated attacks of partial obstruction or a final one of complete obstruction have prompted such investigation. The

following case is fairly typical of the history encountered, the methods of investigation employed to explain it, and the surgical procedure indicated, when cancer originates in the descending colon.

A man, aged 61, always previously well, had no complaint to make until one week before he sought advice. Then he had for the first time abdominal cramps and difficulty in getting his bowels to move, but after castor oil they did move well, and the cramps disappeared. A few days later, however, his pains began again, and this time no home remedies succeeded in overcoming them or in making the bowels move. He had had no bowel evacuation for two days, pains continued, he had vomited frequently and could retain no nourishment. The entire abdomen was distended and tender but more so on the right side than on the left with a prominence in the region of the cæcum that erected itself and became very tense at the time of a cramp. There was greater distension, tenderness and rigidity along the course of the entire ascending colon, apparently, than anywhere else. Temperature, pulse and respirations were all normal. Lavage was done to relieve vomiting, hot turpentine stupes were applied to the abdomen and repeated attempts to induce bowel evacuation were made by various kinds of enemata, such as those described in the chapter on acute obstruction, but no treatment was successful. It was, therefore, decided after three days of such efforts to open and drain the cæcum as a preliminary measure. This gave immediate relief and permitted further investigation to be made with safety. Even after retained gas was thus allowed to escape, no palpable tumor could be discovered. But a barium enema was arrested near the splenic flexure, showing a very narrowed channel there for about 2 cm. along the descending colon, and a barium suspension introduced into the bowel through the cæcostomy filled an apparently normal bowel as far as the upper end of the descending colon, where it was arrested at a site corresponding to the point of constriction demonstrated by the enema from below. Operation revealed a carcinoma at the upper end of

the descending colon exactly where it was shown by the x-ray films. A wide excision was made of the area involved and of the entire splenic flexure as well and communication established between the transverse and descending colon. Pathological examination of the specimen removed proved it was adenocarcinoma. The patient recovered promptly from operation, but recurrence took place six months later and led to the patient's death.

### *Cancer of the Sigmoid Colon*

Beyond the descending colon the incidence of cancer increases, and involvement of the sigmoid and of the recto-sigmoid junction is encountered with distressing frequency, distressing because the growth is commonly so far advanced in these situations before it is discovered that successful removal is most difficult or is soon followed by recurrence. In the sigmoid the first symptoms are commonly those of obstruction of the bowel, appearing suddenly or gradually, with the cramps that result from waves of peristalsis thrown against the barrier. But constipation does not always precede this emergency, and on the contrary diarrhœa may be the chief complaint before obstruction occurs. Blood in the stools becomes a frequent and prominent sign of cancer in this region or farther down in the rectum. Whether it occurs only at intervals or constantly, whether it is profuse or slight, whether the discharged material is dark and clotted or bright red, recurrent bleeding from the bowel should always excite concern and prompt, thorough investigation. It by no means indicates malignant disease of the intestine in every case, but it does so often enough to arouse apprehension, when it forms a part of the patient's history. A tumor may be felt through the abdominal wall but frequently is not, either because it is too small or because it is obscured by a mass of retained fæces above it. By the rectum

it is usually impossible to palpate a tumor of the sigmoid or recto-sigmoid junction because it is far too high and out of reach. But the attempt should always be made, for the weight of such a tumor will sometimes cause it to drag down a loop of the bowel that contains it until it is low enough in the pelvis to be detected by rectal examination even though direct contact with it is impossible. In women such a mass may be discovered more readily by vagina. In this part of the bowel the proctoscope also becomes available for diagnosis and may reveal the presence and character of a tumor. The most satisfactory evidence of all, however, is obtained from x-ray films after a barium enema.

The different character of the symptoms produced by cancer of the sigmoid in different patients, as well as the difficulties encountered in its recognition, are illustrated by the following case histories.

1. A woman, aged 64, had been previously well except for a series of cramps in the bowels, always relieved by getting the intestinal contents well cleared out by a laxative. When she sought advice, after this had gone on for several months, it was because these pains had recurred and persisted off and on in spite of the remedies previously employed. The pain was felt more on the left side of the abdomen than the right. The bowels at this time were constipated but not obstructed. The patient was pale but well-nourished and had lost no weight. No abnormality was found in any organ on physical examination, and the abdomen particularly showed no irregularity in contour, no distension, no palpable mass, no unusual rigidity or tenderness. By following a smooth, non-residue diet and by keeping her bowels regular with a mild laxative, her pains subsided, and she remained apparently well in every way for several months. Then came a sudden complete obstruction of the intestine, necessitating immediate operation for relief. This revealed an annular scirrhus carcinoma of the sigmoid colon. She did not long survive the operation.

2. A woman, aged 60, had been ailing for two years, when she first came under observation. Her illness began with the onset of sudden, severe pain in her left lower abdomen, lasting for two or three days, with the passage by bowel of blood and pus. Subsequently she had similar attacks recurring about once in two weeks, never lasting over four or five days. Between these she was free from pain but felt weak, and her bowels moved too freely every day and night, as many as eight to ten times in twenty-four hours. The stools always contained much mucus, and at times were black for days in succession, and during a severe attack with pain they always contained visible blood, in large black clots resembling liver. She had not lost in weight but much in strength.

Physical examination showed a woman of good color with no emaciation. The abdomen was distended and tympanitic throughout, resistant to palpation, more so on the left side than on the right, and more over the left lower quadrant than the left upper; but no definite tumor was palpable. At the time of examination the stools were formed and showed some blood-stained mucus, but no pus, no ova and no parasites.

This patient left the hospital before the diagnosis was completed and returned to her home in Nevada under the care of her local physician. He reported that she continued to have her attacks of pain as often as once a week, for one or two days, with the passage then of blood in the stools, bright red at first, then black, thick and clotted. The bowels were constantly too loose, never constipated at any time. Finally she returned to the hospital six months after her first admission, because no medical treatment had been of any avail. Operation revealed an annular constriction of the colon at the recto-sigmoid junction. The colon was resected and the growth removed. Pathological examination of the tissue excised showed it to be adenocarcinoma which had almost completely occluded the bowel. The patient did not survive the operation.

In both the foregoing cases, observed a number of years ago, failure to recognize the patient's true condition could have

been obviated by the use of diagnostic methods now employed as a routine. In the first case, x-ray films after a barium enema probably would have revealed the cause of the symptoms, and in the second the proctoscope would have demonstrated at the recto-sigmoid junction the ulcerated tumor that caused the bleeding as well as the diarrhoea and the pain. But whether earlier recognition would have saved life in either case is a question. Even successful removal does not always mean cure, and there is apparently no such thing as early diagnosis of cancer of the sigmoid. In the following case, seen much more recently than either of the two preceding, with every routine facility employed for diagnosis before operation was done, the results were no better.

3. A woman, aged 55, complained of nothing except that "her bowels seemed stopped up". She had a good appetite and had lost no weight. For years past she had been constipated and had to employ laxatives constantly. But now there was greater difficulty than ever before, and for a month past no medicine acted satisfactorily, and no enema seemed to enter the bowel far enough to be of use. Recently she had seen some bloody mucus in her stools and occasionally a blood clot. There had never been any cramps or any severe pain, but occasionally a backache low down. Walking caused a sense of pressure in her rectum, and when she thought she was having a bowel movement, only mucus was expelled.

The abdomen was prominent but tympanitic throughout. There was no mass palpable through its wall. On pelvic examination a large, hard tender tumor was found behind the uterus. While this was best recognized by the vagina, it was also felt high up by the rectum. X-ray examination by fluoroscopy and films showed the barium enema entering readily into the rectum, but there was great delay in filling the descending colon, and it was not until about ten minutes after the rectum was filled that a small streak of barium could be seen passing through the narrowed area into the descending

colon. The latter could be filled gradually as far as the splenic flexure but not beyond because of the persistent obstruction in the sigmoid. The area of narrowing in this part of the bowel shown by the films was about 6 cm. in length and ragged in outline. At operation for cancer of the sigmoid a tumor was found so large and widespread that its removal was impossible. A left colostomy was done. The patient died five months later.

### CANCER OF THE RECTUM

Malignant neoplasm originating in this terminal portion of the large bowel is one of the most insidious and terrible diseases ever encountered. Almost without exception it has progressed beyond the stage where successful removal is possible before it causes symptoms that lead the patient to seek advice. Signs of serious disturbances of health such as anæmia, cachexia, loss of weight and strength never occur early, and local manifestations meanwhile may be so seemingly unimportant that they are entirely overlooked or else misinterpreted. These local evidences of disease are (1) those due to bowel obstruction, or (2) those due to pressure on surrounding parts or (3) those due to ulceration of the neoplasm.

As regards the *first*, difficulty about getting the bowels to move comes on so gradually, when the rectum is involved, and may be overcome for so long a time by the use of cathartics, that the growth is far advanced before increasing occlusion finally compels the patient to seek assistance. Before this stage is reached there may be no pain of consequence and none at all except that attributed usually to "wind". Good appetite continues, and there is no loss of weight or strength. As regards the *second* group of symptoms, those due to pressure by the tumor on its surroundings, they include pain in the lower back and down the legs and bladder irritation. These may be

added to those of persistent and increasing constipation or may be the only ones of which complaint is made. As regards the *third*, the very earliest manifestations may be those due to ulceration of the neoplasm. Then the story is one of chronic diarrhœa, often bloody. There are commonly several loose bowel movements each morning after rising, sometimes with tenesmus, until the rectum is empty, and then no more until the next day. These stools contain mucopurulent material mixed with fæces and not infrequently with bright red blood as well. Occasionally the first symptom that alarms the patient is the passage of bright red blood. Even then the assumption is apt to be that this means nothing but "piles", and only too often the physician also is inclined to accept this explanation without thorough investigation.

As a means of diagnosis, digital exploration of the rectum becomes of the first importance when the tumor is so low in the bowel. For a long time the most careful abdominal palpation may disclose no tumor because it is hidden below the brim of the pelvis, but during all this time palpation from below through the anus may at once reveal the tumor's presence. More than this, abdominal examination may continue to show no abnormality whatever until such time as a tumor has developed large enough to be felt in the inguinal region, or the liver has become enlarged by metastases, or masses of enlarged glands can be palpated, or ascites has resulted as a consequence of generalized involvement of the peritoneum, and yet long before these late developments have taken place, comparatively simple examination of the rectum by the index finger will at once detect the mass that obstructs the bowel, that presses upon the sacrum behind and the bladder in front, and that bleeds because it has become necrotic.

If such a mass is not easily reached when the patient is lying down, it may be when standing up and leaning forward over a chair or a table, or in a squatting posture, or when one hand over the abdomen pushes down from above while the index finger of the other hand presses up from below through the rectum. But if every such device fails to make the tumor palpable, it may still be easily discovered by the proctoscope, when it involves the rectum. A routine rectal examination in every case of chronic digestive disturbance, of chronic constipation or diarrhœa, or chronic backache and particularly of chronic bleeding from the bowel, will sometimes save the diagnostician from grievous error, and the patient from fatal delay. The tendency too often is to forget this method of investigation, as Osler used to impress upon his students forty years ago. Even x-ray films after a barium enema are less important as a means of diagnosis of rectal cancer than direct exploration by the finger supplemented by the proctoscope.

But every tumor found involving the rectum does not necessarily mean cancer. *Hæmorrhoids* may really be responsible for the sacral backache, the constipation or diarrhœa, the bleeding from the bowel and a progressive anæmia, as well as for a palpable mass found by digital exploration. In these cases the proctoscope will reveal the real lesion more accurately than any other method of investigation. Unfortunately the knowledge that "piles" can cause recurrent bleeding from the rectum, interference with bowel function and backache, too often deludes patients into postponing examination too long.

The *uterus* in women and the *prostate* in men may be the site of the tumor palpated by rectum and not the bowel at all. Vaginal examination by bimanual palpation and by speculum will make clear the fact that the tumor originates in the uterus,

and careful palpation by rectum in men supplemented by cystoscopy and urinalysis will identify the prostate as the site of the tumor. In women this may be due to a fibroid and in men to simple hypertrophy with no malignancy at all. But cancer originating in the uterus or the prostate may invade the bowel wall by direct extension, so that the neoplasm involves both structures, and its removal by operation becomes impossible or at least futile.

*Diverticulitis* may cause a tumor palpable by rectum, but usually this lies too high to be recognized in this way or even by the proctoscope, involving the sigmoid colon rather than the recto-sigmoid junction or the rectum itself. It is most readily identified by x-ray films after a barium enema. There is always a possibility that a tumor of the rectum discovered by digital exploration may be a granuloma due to *syphilis*, *tuberculosis* or even chronic *amæbic infection*, but the possibility is so remote that it is better to remove the tumor first and determine its character afterward by microscopical examination of the tissues excised. There is always a possibility also that the symptoms suggesting cancer of the rectum, when no tumor is palpable, may be proved by the proctoscope to be due to *intestinal polyps*. Their discovery and removal may prevent cancer of the bowel later on. Finally a tumor may be palpated high up in the rectum as a hard ridge about the level of the upper border of the sacrum, due to metastatic deposit there and not to primary rectal carcinoma. This so-called *rectal shelf* usually is suggestive of cancer higher up in the digestive tract, in stomach, liver, pancreas or colon.

Illustrative cases of cancer of the rectum are altogether too numerous, and their history has a monotonous sameness as regards outcome, even though the evidence varies somewhat

upon which diagnosis is based. There are facts in the clinical history, however, as regards the patient's age, the duration and character of the symptoms and the clinical course better impressed by recounting individual instances than by didactic general statements. The following case is of special interest because it shows the early age at which cancer of the rectum may occur as well as the fact that the first manifestation may be hemorrhage. The frequency of bleeding is shown by all these cases.

1. A man, aged 30, complained that for months he had noticed at times a little bright red blood in his stools. But a few days before he sought advice he had passed a quantity of bright red blood with some clots, and after that more or less bleeding had persisted every day. He was a large well-nourished man, and no abnormality whatever was found in his abdomen except that he was tender low down in his left inguinal region. But digital examination by rectum showed a tumor obstructing the bowel, irregular in contour, only a short distance above the anus. Operation was advised but refused. Instead, he consulted a Chinese doctor to be cured by herbs. When seen again six months later his rectum was completely obstructed by a large tumor mass practically filling the entire pelvis, irregular, nodular, tender with bloody, purulent discharge from the anus. Nothing could be done then except a colostomy. A portion of the growth removed at that time for biopsy showed it was an adenocarcinoma.

2. A woman, aged 75, stated that as long as five years previous she began to notice clots of blood in her bowel movements and at times bright red blood. This had continued at intervals ever since. At the time she finally sought advice, she was passing bright red blood every day, even without a bowel movement, and had to wear a cloth always, even at night, because pus and blood ran constantly from her bowel. Besides this she had pain in the rectum now and then, a sense of pressure and fulness there most of the time and suffering particularly when at stool. The bowels were obstinately constipated, and while physic would cause them to move, it pro-

duced such intense pain and tenesmus that she preferred to avoid it. She had lost fifty pounds in two years. Rectal examination showed an irregular, hard mass the size of a turkey's egg about three inches from the anus with soft necrotic edges. Operation was declined. Six months later, before she died, the mass completely filled the pelvis.

3. A man, aged 74, previously well, had been troubled for three months by frequent loose bowel movements every day and a constant feeling of fulness and pressure in his rectum, as if there was something retained that ought to be expelled. This was always worse in the morning, when he had a series of bowel movements with gas, chunks of solid material and quantities of fluid that came with an explosion. He also passed blood in his stools quite frequently, which he attributed to "piles". He had no real pain but had lost much in weight and strength. Digital examination revealed at once a hard mass in the rectum, only a short distance above the anus, that bled easily, obstructing but not completely occluding the bowel. Operation was advised. The neoplasm was removed, but the patient died one week later.

4. A man, aged 49, presented himself complaining of a bloody discharge from his bowels. For two or three years he had noted occasional bleeding after a bowel movement, which he thought meant piles and disregarded. But for three months before he sought advice, nearly every passage had shown bloody mucus, though the stools usually were formed. There was no constipation, but on the contrary two to five defecations every day. No pain preceded or followed. Digital examination by rectum at once disclosed a hard palpable mass, easily within reach. The proctoscope confirmed the presence of this tumor and a bit of tissue removed for biopsy proved it was adenocarcinoma. Radical removal was done at once, but the patient died soon afterward.

### TREATMENT OF CANCER OF THE INTESTINE

For cancer of the intestine, no matter in what part it occurs, there is no cure possible but surgery. The best advice

that can be offered the patient is excision of the growth as soon as possible after diagnosis has been made. But in many instances this is already impossible, when the patient is first seen, because of the wide extent of the involvement or of metastatic transfer of the disease to other organs, particularly the liver, before the condition is discovered. Then a colostomy has to be substituted for excision, the making and maintaining of an opening through the abdominal wall into the bowel to allow the discharge of fæcal matter in spite of the obstruction farther along in the natural channel. This operation, of course, is not curative but merely prolongs life. If a successful removal is made of the neoplasm, and the patient apparently recovers good health, there is always the danger of recurrence, which all too often takes place within a few months or a year. When only a colostomy is done, or when there is a disappointing recurrence after a successful operation for removal, it requires all the wisdom and tact imaginable on the part of the physician to give the patient comfort and to maintain courage and morale. As with inoperable or recurrent cancer of the stomach, the fight is carried on against insurmountable odds with the consciousness that ultimate defeat is inevitable. Every case demands its own management, and there are no rules for treatment applicable to all. Such soft and liquid foods should be given as create the least disturbance with digestive agents as required, stimulants of all kinds to meet increasing weakness, and opium in some form to overcome pain. But above all there is needed a cheerful philosophic attitude of mind that takes into account the patient's mental and spiritual as well as his physical problems.

## CHAPTER XXV

### POLYPS OF THE INTESTINE

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The significance of intestinal polyps lies in the fact that the symptoms they produce, pain in the bowels, diarrhœa, blood in the stools and at times obstruction, resemble those of cancer of the colon, and in the further fact that polyps benign at the outset have a tendency to become malignant as time goes on. That polyps form in the intestinal wall is no new discovery, for they have long been found at autopsy and in recent years by the proctoscope during life. But the ability to diagnose their presence in the living subject, the knowledge of their frequency, their extent in the bowel, the symptoms they produce, their menace to the patient and their proper management have all been acquired largely during the past decade.

#### INCIDENCE, TYPE AND CLINICAL HISTORY

Polyps occur in the small intestine, but only rarely as compared with their incidence in the large. In the lower colon a single polyp may be found, or there may be several in a group, or so many that the entire rectum and colon is studded with with all types of flat and pedunculated adenomata, large and small, presenting the condition known as *multiple polyposis* or

*diffuse adenomatosis*. But such a condition is far from common, and usually the number observed is small. In form intestinal polyps are most often pedunculated, but they may be sessile. In size they vary greatly, from mere tags of the mucosa to tumors 3 to 4 cm. in diameter and 2 to 10 cm. in length.

Considerable controversy has arisen about the origin of such polyps, whether they are congenital or acquired. It seems probable they are never congenital, because, as Bagen points out, there is no case on record where polyposis has been found in the intestine at birth, and they are seen with increasing frequency as age advances. But there is undoubtedly an inherited tendency to their formation, for cases have been reported where several members of the same family were afflicted, suggesting that some weakness of the bowel wall is transmitted that permits polyps to develop more readily than in the normal intestine. Of the acquired cases, two groups are recognized, one post-inflammatory, following chronic ulcerative or amœbic colitis, where polypoid excrescences result from hyperplasia at the site of former ulcers, since healed, and another group of true adenomata without any history of previous inflammatory disease. The first group Bagen (Proceedings of the Staff Meetings of the Mayo Clinic, Oct. 9, 1935.) classifies as pseudo-polyps, the second as true polyps, and further distinguishes between them by the fact that pseudo-polyps usually are benign, though rarely they may become malignant, while true polyps become malignant in the great majority of cases. Patients with multiple polyposis are much more likely to develop cancer of the colon than patients with only one or a few of these growths. Nevertheless multiple polyps are found in the colon occasionally at autopsy, where they have existed for an unknown time without undergoing malignant change.

The symptoms produced by intestinal polyps are indefinite. There may be none at all, so that the discovery of their presence is made only at autopsy or at operation for some other disease. But the symptoms usually described are increased frequency of bowel movements and the occasional passage of blood. They do not cause pain unless they obstruct the bowel, but sometimes they attain a size so great that they do interfere with the bowel lumen, even without malignant change. Then cramps accompany the diarrhoea and become a part of the clinical history. The story that suggests intestinal polyps is, therefore, even more likely to suggest carcinoma of the bowel, and the latter is a much more frequent explanation than the former.

#### RECTAL EXAMINATION

External examination of the abdomen reveals no abnormality unless a tumor is palpable, and then there is more than the presence of polyps to account for it, though polyps may also be present, and one of them may have formed the basis on which neoplasm developed. It is by internal examination, however, through the rectum, that diagnosis is made, if at all. Digital exploration may discover, if polyps are low enough in the bowel, a number of protrusions from the surface that are quite different from the feeling of the normal mucous membrane. What these are, however, is proved with certainty only by the proctoscope. In most cases polyposis involves the rectum and sigmoid colon, whether the condition extends higher in the bowel or not, and thus the diagnosis can be made by direct inspection, though the extent of the disease may remain undetermined by this method of investigation. If no polyps are seen through the proctoscope in the terminal 18 cm. of the

bowel, probably none exist, but exceptionally there may be a few higher up beyond the range of the instrument. Such direct inspection gives information about the size and character of the polyps as well as about their presence and their number and permits removal of a portion of tissue for biopsy, if there is a suspicion of malignancy.

### X-RAY EXAMINATION

By special technique developed in recent years it has become possible to demonstrate in x-ray films polyposis of the colon. This requires first a careful emptying of the bowel by two ounces of castor oil the night before, followed the next morning by a cleansing enema of normal salt solution. Then with the patient fasting the barium mixture is introduced into the colon and films are taken. After an hour or two interval, during which the patient expels as much of the barium enema as possible, the bowel is inflated and distended by gas. Air as a rule proves satisfactory for this purpose, but oxygen or carbonic acid gas may be employed instead. Then further films are made, and if polyps are present, they stand out prominently because coated over still by a layer of barium attached to their surface.

### DIAGNOSIS

A history of chronic diarrhoea with the occasional passage of blood and with cramps in some instances suggests the possibility of cancer of the colon and prompts special examination, but does not by itself contain any details that identify polyposis as the cause. By digital exploration of the rectum the polyps may be palpated, but this permits only a limited area of the bowel to be investigated. By the proctoscope and

sigmoidoscope further information is obtained as to the extent of the polyposis, as to the character of individual polyps, and as to whether malignant degeneration of one or several has developed. But even so the region that can be explored in this way is limited to the lower 18 cm., while polyps may involve any part of the colon. To determine whether they are present beyond the point that can be directly inspected, x-ray films are required with special technique such as has been described in the previous section.

### TREATMENT

When only small polyps are found in the rectum or sigmoid colon following a history of ulcerative colitis or chronic amoebic infection, even these pseudo-polyps or mucosal tags should be destroyed so far as possible by fulguration through the sigmoidoscope, for according to Bargaen approximately 25 per cent. of such patients develop carcinoma later on. When there is but a single adenomatous polyp, 5 per cent., according to Brust and Buie (Proceedings of Staff Meetings of the Mayo Clinic, Oct. 17, 1934.) develop cancer of the colon on this as a base. Such a polyp should, therefore, be excised without waiting for possible malignant change in it. In the cases of multiple polyposis, where hundreds of such adenomata can be counted, the chances of escaping carcinoma are infinitesimal, according to Brust and Buie, and the probability that cancer will develop in one or several of these is nearly 100 per cent. In such patients wide excision of the polyp-bearing area of the colon is advised even to the extent of complete colectomy with destruction by fulguration of any remaining polyps in the rectum below the site of operation.

## CHAPTER XXVI

### ACUTE INTESTINAL OBSTRUCTION

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Of all the functions of the intestinal tract the most important and most vitally essential one is to provide a passage for the regular elimination of waste from the body. If this is suddenly stopped, disaster is certain to follow quickly, and unless some means of relief is afforded promptly, death is inevitable. Acute intestinal obstruction is one of the emergencies of life that come as a rule without adequate warning, thrusting the patient within a few hours from apparently good health into a situation where the end of existence is imminent. It, therefore, demands recognition without loss of time and immediate action.

#### CLINICAL HISTORY

There are a number of different ways in which acute intestinal obstruction may be produced, but no matter what has brought it about, there are certain effects common to all. These are inability to make the bowels move, pain, vomiting and tympanitic distension. The onset may be sudden, “out of a clear sky”, or preceded by a number of minor and tem-

porary attacks of partial obstruction. The first warning usually is colicky pain that prompts desire to empty the bowel. Attempts to accomplish this, however, are futile, except that at first material may be extruded that was already below the site of obstruction before the contents above it were blocked. In spite of this defecation, however, no relief from pain follows. Waves of peristalsis continue to be sent against the obstacle in the effort to overcome it and so the cramps persist. When the bowels fail to move again, the patient usually resorts to some of the common purgatives, such as castor oil or Epsom salts. These, however, only increase the violence of the pain by adding to the force of the peristaltic waves without being able to force the bowel contents past the obstruction. Enemata of various sorts likewise prove unsuccessful. No bowel movement can be obtained. The pains continue. The next evidence of trouble is nausea and vomiting, partly due to reverse peristalsis, partly to toxæmia. Gradually no medicines, no food, not even water can be retained. Vomiting persists, even though nothing is swallowed, and the material thus evacuated little by little assumes a character suggesting by its appearance and odor that of intestinal contents. Then finally the abdomen begins to swell and to become more and more distended by gas. This stage should never be awaited before a diagnosis is made, but it is usually the patient who delays about calling the physician until the condition has progressed as described. Attempts to remedy the difficulty are made first at home by the sufferer and the family, the seriousness of the situation is not recognized promptly, and it is not until after the ordinary measures adopted have failed that the patient becomes alarmed. The story as related has numerous variations and modifications, depending upon the site and nature of

the obstruction, but these will be considered as each separate form of obstruction is surveyed. The general features portrayed are those common to all.

### PHYSICAL EXAMINATION

Here again there are certain characteristic signs produced no matter what has caused intestinal obstruction and that are seen in all forms. Variations arise, according to the site where the bowel has been blocked and the nature of the accident that caused it, but in every case alteration of the normal abdominal contour appears promptly. There may be a general symmetrical distension, not more noticeable in one region than in another, or a localized bulging limited to one certain area, where a particular coil of intestine has become distended by gas. There may be noted also a visible change in this bulging area, so that it increases or decreases in prominence as a peristaltic wave runs over it. Such erections of the bowel wall occur following or accompanying a severe colicky pain and subside again as the cramp disappears. They may be discovered by palpation even when not visible on inspection. Tenderness is not likely to be extreme or generalized, but is often found over some part of the abdomen that marks the site of obstruction or the bowel coil that is distended behind it. Only in exceptional instances can a tumor be discovered, in the first place because acute intestinal obstruction is only occasionally due to a tumor, and in the second place because the accumulation of gas that results soon obscures a tumor even if one is responsible. Percussion gives a tympanitic note of equal intensity throughout or else limited especially to some part of the abdomen where obstruction has caused localized distension. Finally, auscultation should be employed as

routinely in examining the abdomen as in examining the chest, for the stethoscope will elicit sounds that prove active peristalsis is going on in the bowel or none at all, if the intestinal wall is paralyzed, and this differentiation often is vital to diagnosis.

Attention should be directed during the examination especially to certain features that aid in explaining the nature of the obstruction. First, in every case careful investigation should be made of all points at which hernia commonly occurs, such as each inguinal and femoral region, the umbilicus or the scar of a former operation, for hernia with strangulation of a protruding knuckle of bowel is an occasional cause of acute obstruction. Second, scars in the abdominal wall, indicating a previous laparotomy, are of significance not simply because they form a possible site for hernia, but also because they mean that post-operative adhesions may be the cause of obstruction. Third, digital examination by rectum in either sex and by vagina in women may give valuable information about conditions in the lower abdomen that may escape notice by palpation from outside.

#### LABORATORY EXAMINATIONS

The most significant fact about the *stools* is the absence from them of any real *fæces*. There may be a little *fæcal* matter passed at the outset of the attack, soon after obstruction has occurred, coming from the bowel below the point where its lumen is no longer patulous, but none is seen after the lower bowel is empty. Later discharges in response to enemata usually are nothing but blood-stained mucus or almost pure blood. The character of the material evacuated may give a suggestion as to the nature of the obstruction, but the most

striking observation on stool examination is the absence of normal bowel contents. *Stomach contents*, obtained soon after the emergency has arisen that calls for investigation, may contain food eaten at the last meal previously. But later on they become a brownish dark fluid, foul in appearance and odor, resembling the material found in post-operative acute dilatation of the stomach. Frequently the odor is distinctly faecal, because they really consist of intestinal contents from above the site of obstruction, forced into the stomach by reverse peristalsis. But this development is not essential to diagnosis and should not be awaited in order to establish it. The *urine* usually is scanty, and its secretion may be suppressed completely by shock and toxæmia. This should not be misinterpreted, for it is the function and not the structure of the kidneys that has been affected. The presence or absence of indican in the urine is no longer considered of importance in the diagnosis of intestinal obstruction. The *blood count* shows a gradual increase in leucocytes as obstruction persists, due to inflammation about the latter's site, and the number of white corpuscles may rise as high as 20,000 or above. But in some forms of obstruction this may not occur at all, and when it does, it is never characteristic of obstruction only but may be due to any acute inflammation in the intestinal wall.

### DIAGNOSIS

Every patient, complaining of failure to get the bowels to move according to their usual habit, may have the first stage of intestinal obstruction and deserves careful observation, even though at the outset examination reveals little or nothing abnormal. Once the definite clinical picture is presented that suggests acute intestinal obstruction, there are still many un-

certainties as to the attack's origin and course. The first problem is to determine that the symptoms and signs are really due to mechanical obstruction and not to some other pathological change that simulates it; and the second problem is to discover if possible the site and character of the obstruction.

*Acute General Peritonitis.* — There are several other conditions that may produce similar symptoms and signs but the one that most often leads to confusion in diagnosis is *acute general peritonitis*. This likewise begins with colicky pains in the abdomen; quickly causes complete interruption of peristalsis and of bowel discharges by paralysis of the bowel wall; gives rise to persistent vomiting of bad-smelling stomach contents even when no food or drink or drugs are taken; and ultimately leads to general and excessive tympanitic distension, with tenderness, fever and extreme prostration. There are points in the history, however, that help to distinguish this variety of disaster from the other, particularly the time that the symptoms take for their development and certain preliminary incidents that lead up to the catastrophe as finally observed. Acute general peritonitis usually requires several days or even a week to reach the complete arrest of bowel discharges, the degree of tympanitic distension and the evidences of profound toxæmia that result within twenty-four or forty-eight hours from sudden mechanical obstruction. During this period of more or less gradual development or at least preceding its extreme degree as ultimately observed, there are manifestations of other disease conditions that anticipate and make possible the acute general peritonitis. Such antecedent events are a ruptured appendix as a result of acute appendicitis, a ruptured or perforated or infected uterus as a conse-

quence of induced abortion, puerperal infection following an apparently normal childbirth, rupture of a pelvic abscess originating in a Fallopian tube, ruptured empyema of the gall-bladder, perforated gastric or duodenal ulcer, perforated intestinal ulcer caused by typhoid fever, tuberculosis or cancer, or recent abdominal operation with subsequent infection. Each of these accidents should have its own particular story leading up to the symptoms and signs of the ultimate predicament in which the patient is seen for diagnosis. But unfortunately such an illuminating history can not always be obtained. The patient is mentally unfitted by fever and toxæmia to make a clear and rational statement of the course of events. The family and friends are too much perturbed by the obvious peril of the situation to give an intelligent explanation of what has been going on, and only too often, in the case of an abortion, deception is practiced deliberately in order to cover up some one's criminal interference. Therefore history is after all a fallacious guide, and too much dependence can not be placed upon it in determining that the case is one of general peritonitis rather than mechanical bowel obstruction.

Physical examination gives much more reliable information to aid in the differentiation. (*a*) The abdomen in general peritonitis on inspection is greatly distended, symmetrical and smooth; while in acute obstruction it is usually not so excessively enlarged, is apt to bulge in one part more than another, or to show the outline of intestinal coils or loops, producing patterns of various sorts. The discovery of such irregularities in the contour of the abdominal wall can be accepted as definite proof of acute intestinal obstruction, but their absence from the picture does not prove that the case is one of acute general peritonitis nor exclude a mechanical origin for the distension.

(b) Equally significant is the observation of peristaltic waves in the bowel, manifested as a stiffening or bulging of the coil whose outline is seen through the abdominal wall. These so-called erections of the bowel occur following or accompanying a severe colicky pain or cramp, and subside again as the pain disappears. No such sign is ever seen in acute general peritonitis because the intestinal wall is paralyzed by the inflammation of its external coat and by the general toxæmia, so that it has lost temporarily its peristaltic power. Usually, therefore, these visible erections signify the presence of an obstruction against which peristaltic waves are sent in the effort to overcome it. But they are not always seen even when obstruction exists, because the early onset of excessive tympanites may obscure all peristaltic waves. Sometimes these changes in abdominal contour may be observed early in the illness and then disappear entirely. This is ordinarily assumed to mean that in addition to the original mechanical obstruction, peritonitis has developed as a sequel. But this is not always true, because excessive distension of the bowel wall by gas, as the occlusion persists, may result in its ultimate paralysis and complete loss of peristaltic power, even though no secondary peritonitis has occurred.

(c) The distended abdomen of acute general peritonitis is exquisitely tender throughout. The patient resents the lightest touch on any part. Even the weight of the bedclothes is distressing. In acute obstruction tenderness is more often localized to some part where the trouble began and where the blocking of the bowel has occurred. It is not so general or widespread, nor is it so extreme that careful palpation can not be tolerated.

(d) There is a characteristic rigidity or board-like hardness of the abdomen in peritonitis, that is the result of protective

muscle spasm. This is not so likely to be found in acute obstruction except over one area where the bowel is involved, and its discovery at that point and not at others helps to identify the site of the difficulty as well as its character.

(e) Palpation of a tumor in the abdomen is much more likely to mean obstruction than peritonitis, but except in certain forms of obstruction, such as that due to intussusception or neoplasm or tuberculosis, no tumor is ever palpable, and even though one exists, it is usually obscured quite early by the resulting tympanites. On the other hand, a tumor mass discovered early may really be the underlying factor that has formed the point of beginning for an acute general peritonitis, such as an appendix abscess that ruptures, or a cancer or tuberculosis of the bowel wall that leads to its perforation.

(f) Examination by the vagina may reveal the condition of uterus, tube, or parametral tissues that preceded a general peritonitis caused by rupture of an abscess or by direct extension of infection, or the bulging of the vaginal vault, fixation of the uterus and general rigidity and tenderness that characterizes pelvic peritonitis, showing where the conflagration originated before it spread to the general peritoneal cavity. In the case of mechanical obstruction of the bowel, vaginal examination shows no abnormality at all, or possibly a distended knuckle of bowel, not particularly tender.

(g) Palpation by rectum may discover a tumor mass, fæcal or neoplastic, that is causing obstruction, or the intussuscepted bowel crowded down far enough to be within reach, or it may reveal no abnormality at all. In the case of acute general peritonitis, usually no evidence can be obtained from rectal examination; but negative findings do not exclude acute intestinal obstruction.

(*h*) Palpation of all orifices where hernia might occur sometimes results in the finding of a knuckle of bowel caught and strangled, at the navel or over one or the other inguinal ring or femoral ring. Thus not only the fact but the character of an acute intestinal obstruction will be demonstrated, and acute general peritonitis will be excluded.

(*i*) When percussion of the abdomen shows everywhere a high-pitched tympanitic note, indicating excessive distension of the bowels by gas, the condition is much more likely to be acute general peritonitis than acute intestinal obstruction. The tension is sometimes so extreme that the splenic dulness is obliterated, the liver dulness decreased and even the heart pushed upwards. In acute intestinal obstruction localized tympanites is found over the distended bowel above the site of occlusion, due to accumulation of gas that can not escape. If the point where the block has occurred is low in the colon, tympanites may be found widespread over the abdomen, but even so it is rarely so excessive as in peritonitis, nor does it encroach so frequently on adjacent solid viscera, obscuring their proper area of percussion dulness.

(*j*) By auscultation over the abdomen it can be determined whether peristaltic contractions still go on, or whether the bowel has lost its power. Hisses, squeaks, rumbling and gurgling sounds mean that even though the intestine is obstructed, its wall is not paralyzed, while complete silence is ominous because it indicates the loss of all effort to pass on gas along its natural course, and this always suggests acute general peritonitis.

In spite of all the distinctions enumerated between acute intestinal obstruction and acute general peritonitis, the fact remains that the differentiation is not easy. Face to face with the problem at the bedside, familiar with all the theoretical

points that should distinguish one condition from the other, the physician finds occasionally that the conclusion reached is erroneous, as is shown ultimately by operation or by autopsy. Wrong diagnoses are notoriously frequent in the solution of this particular problem, and no one realizes this so well as the man who has had experience with it and has watched his own well-reasoned conception checked up by the facts in the operating room or on the post-mortem table. Every effort should be made by every method of examination to reach an accurate deduction as to which form of disease is present, but a spirit of humility is much more fitting than one of pride in this uncertain field of diagnosis.

Compared with acute general peritonitis, all other pathological conditions that simulate acute intestinal obstruction are rare. But there are two that haunt the scene in every case. These are acute hemorrhagic pancreatitis and embolus of the superior mesenteric artery.

*Acute hæmorrhagic pancreatitis* presents certain features supposed to identify it but all are unreliable. The disease occurs in obese patients, usually in middle age. Frequently a history of typical attacks of gallstone colic precedes. Then comes a sudden paroxysm of greater severity than any that have gone before with intense pains in the upper abdomen, followed by vomiting frequently repeated, and in a large percentage of the cases by complete cessation of all bowel movements, which leads to the erroneous diagnosis of intestinal obstruction. As in the latter accident, there is no fever at the outset, but the pulse is rapid and the patient in a state of shock. Tenderness is found on palpation over the upper abdomen and resistance to deep pressure but no palpable mass. The patient's condition is extremely serious from the beginning and runs a rapid course to a fatal

termination within three or four days or a week. Most often the diagnosis fails to be made because this possibility is forgotten, and the patient is supposed to have acute intestinal obstruction until operation or autopsy reveals the real pathology. Early operation affords the only hope for cure, and fortunately this is indicated for either of the conditions under consideration. Therefore, if the slightest doubt exists, exploratory laparotomy is the proper procedure not only as a means of diagnosis but also because it is the best method of treatment.

*Embolism of the superior mesenteric artery* likewise makes its onset with sudden severe pain in the abdomen, colicky in character, followed by persistent vomiting, rapid pulse, abdominal distension and all the signs of shock. It is significant that in the beginning the bowels may be stimulated to increased activity so that diarrhoea results with bloody discharges, but this soon is replaced by the extreme constipation and rapidly increasing distension of the abdomen that suggest acute intestinal obstruction. This fact, that early diarrhoea with bloody stools precedes the obvious obstruction that occurs shortly afterwards, would be more valuable in differential diagnosis, if it did not form a part in the history also of intussusception. Another aid in recognizing that embolism has occurred is a history and physical signs of endocarditis. But in any event, as in acute hæmorrhagic pancreatitis, exploratory operation is indicated as offering the only hope for cure. For as a result of interference by the embolus with the blood supply to the intestinal wall, hæmorrhagic infarction follows, the affected portion of the bowel dies, and it must be resected if the patient's life is to be saved.

Once it has been determined with reasonable certainty that the patient has an acute intestinal obstruction and that the symptoms and signs are due to this and not to some other

disease, the second problem presenting itself for solution is this: What is the character and site of the mechanical obstruction?

*Adhesions.* — After every abdominal operation probably more or less adhesions are formed, fastening coils of intestine to each other or to the abdominal wall. Yet singularly little trouble results from this as a rule, and it is only exceptionally that there is any serious consequence. Once in a while direct compression of the bowel by such an adhesion in the form of a thin band, or fixation of it in a way that causes angulation or kinking, leads to obstruction so that intestinal contents can no longer pass. This may occur soon after a laparotomy or not for years after, even though the adhesions have been present ever since the abdomen was opened and its contents handled. Such adhesions may form at times even without any previous operation to account for them, as a consequence of some forgotten inflammatory attack. Minor interferences with the bowel's motility may precede complete obstruction, or the acute attack may appear without warning, and nothing in the patient's previous history may be found to explain its origin. Then dependence for diagnosis must be placed first of all upon the character of the symptoms. Obstruction by adhesions usually involves the small intestine rather than the large, and the effects are manifested more in the upper than in the lower abdomen. There is of course immediate stoppage of bowel evacuation, though there may be one defecation even after blocking has occurred, with the discharge of material from below the site of obstruction, passed voluntarily or after an enema. Sudden onset of pain, followed by repeated paroxysms as violent peristaltic waves attempt to overcome the obstruction, early nausea and vomiting with the vomitus consisting first of stomach contents, then gradually of intestinal contents

regurgitated into the stomach by reverse peristalsis, recognized by their appearance and their fæcal odor, rapid development of shock and toxæmia manifested by rapid pulse, cold and clammy skin, sunken features, profuse perspiration and extreme weakness and in some cases persistent hiccoughs as a most distressing feature; these are the symptoms of acute intestinal obstruction produced by adhesions. On physical examination the distension found, gradually increasing in degree, is more pronounced in the upper and central than in the lower portion of the abdomen, and peristaltic waves with shifting outlines of intestinal coils are observed by inspection, by palpation and by auscultation, before distension becomes so great that the bowel wall is paralyzed by it. Thus the diagnosis of acute obstruction involving the small intestine is made by the combination of cramp-like pains, early and persistent vomiting soon becoming fæcal, the inability to expel fæces or gas by bowel, the distension most marked in the upper and central abdomen and the visible peristalsis. All this, however, fails to identify the exact type of obstruction, which must be inferred from previous history, the presence of a laparotomy scar and the fact that adhesions constitute the most common cause. After all, the only absolutely certain diagnosis is that made with the abdomen open at operation or at autopsy.

*Strangulated Hernia.* — This is a close contender with adhesions for that “bad eminence” of first place as the most frequent cause of intestinal obstruction. Hernia is a common ailment, but occlusion of the bowel as a consequence does not often happen. But if a knuckle of intestine protruding through an opening in the abdominal wall, at the navel, at either inguinal or femoral ring or at the site of an operative scar, becomes caught so that it is strangled, the same obstruction

of the lumen occurs as by a compressing or kinking band inside the abdomen, and in addition the blood vessels supplying this part are also occluded so that the danger of gangrene is added unless prompt relief is afforded. There may be a history of a rupture, long existent, which has never before caused serious trouble because supported by a truss, but which following some unusual effort has permitted the bowel to be forced out in spite of the truss to such an extent that it becomes caught and retained and can no longer transmit its contents. Or it may be that the first time the abdominal wall gives way and allows a portion of intestine to protrude, the knuckle of bowel may become caught and squeezed in such a way as to cause complete obstruction. Careful search of the various points at which rupture commonly takes place will reveal the tender tumor formed by the incarcerated portion of bowel. The symptoms and signs of acute obstruction produced in this way usually are much the same as when the cause has been intra-abdominal adhesions, because again it is the small intestine that gets caught as a rule in a hernial opening. But there is always the added danger in this case that comes from shutting off the blood supply from the part of the bowel obstructed, and this demands early correction if gangrene is not to follow even though obstruction is relieved.

A woman, aged 71, who previously had never been ill, complained that everything she ate had hurt her stomach for about a month. She wanted food but was afraid to eat because of the suffering that followed. There was no nausea or vomiting but considerable belching of gas. She had lost weight and grown weaker. She herself attributed all of her trouble to worry about a husband, who was a victim of chronic alcoholism. On physical examination no abnormality could be found anywhere to explain her complaint, but she was very tender over the epigastrium. She refused gastric analysis and

x-ray examination. On a liquid and soft diet and rest she improved but never entirely lost her pain after food. Six weeks later she had a sudden attack, characterized by cramps in her upper abdomen, loss of appetite, nausea and vomiting and inability to make her bowels move, though previously they had always been regular. All these symptoms persisted for twenty-four hours in spite of all sorts of treatment including stupes and enemata, and the abdomen gradually became distended. At this time the patient called attention to a lump in her right inguinal region that appeared suddenly a short time before her attack and had never disappeared since, though it was not painful or tender. At operation recommended for acute intestinal obstruction, the obvious hernia was repaired first, as the probable cause, but as it showed no evidence of constricting the bowel and was apparently in no way connected with the patient's symptoms, the abdomen was opened through the mid-line. Then a constricting band was found obstructing the small intestine. Release of this was followed promptly by recovery and the patient remained free from all digestive disturbance subsequently.

*Intussusception.* — An entirely different type of obstruction is that produced by the telescoping of one portion of the bowel down inside the part beyond it, an accident known as intussusception. This is much more common in infants and young children and rarely occurs in adults. It is preceded and induced as a rule by diarrhoea and abnormally violent peristalsis. It involves most frequently the lower end of the ileum, which passes through the ileocæcal valve into the cæcum, but it may occur instead beyond the cæcum, in the colon. Besides the characteristic symptoms of any acute intestinal obstruction, the features that suggest this form, in addition to the age of the patient, are bloody stools, the constant desire for evacuation with straining effort, and a palpable sausage-like tumor discovered most often in the right lower quadrant or else toward the centre of the abdomen. The tumor sometimes can be felt

by rectum when intussusception has occurred low in the colon. The peculiarities described usually suffice to permit this form of obstruction to be diagnosed with accuracy.

*Volvulus*. — A fourth type of obstruction is that produced by a twist of the intestinal tube on its axis, to which is given the name *volvulus*. By such a twist there is produced not only mechanical obstruction but also stoppage of the circulation through this part of the bowel as in strangulated hernia. This accident is not common and probably would never occur if the mesentery were always normal. But sometimes the mesentery is too long, and thus the intestine is given too much freedom so that a twist becomes possible. This form of obstruction occurs most often in the colon and most frequently of all in the sigmoid portion. Therefore, the first localizing evidences, sudden violent pain, visible distension and a tympanitic tumor, usually are found in the left lower abdomen. But the distension rapidly becomes general, the whole abdomen is blown up by retained gas, and soon it is impossible to distinguish this form of obstruction from that caused in other ways. In fact there are no diagnostic signs of *volvulus*, and in most cases it can not be identified positively but only suspected as the cause of the disaster. But unless operation is performed early, the bowel becomes gangrenous from absence of proper blood supply, and peritonitis quickly causes a fatal outcome. Thus, while *volvulus* is a rare, it is a most dangerous form of acute intestinal obstruction.

*Neoplasm*. — The fifth possible way in which obstruction may occur is by a neoplasm in the bowel wall, which forms a tumor mass of sufficient size actually to obliterate the lumen of the intestinal tube, or else by its weight or pressure, after it reaches a certain size, induce kinking. Complete obstruction

may come suddenly without previous warning, or after a series of minor attacks of partial obstruction, relieved spontaneously or by cathartics. The symptoms caused by cancer of the intestine, before it reaches the stage of complete obstruction, and the manifestations by which it may be recognized as the actual cause of the catastrophe, have already been discussed in the chapter devoted to this subject and illustrative cases presented.

*Intestinal Contents.* — Finally, one other way in which obstruction may be produced is by the plugging of the intestinal pipe by its contents. These may be hardened and long retained fæces, or a large gallstone, or a mass of round worms, but only rarely is the blockade due to some foreign body swallowed accidentally, such as a plum or peach stone, or a coin, or false teeth or a marble. Obstruction of this type may take place anywhere in the course of the intestine, but most often happens at one of the points in the large bowel where it makes a bend, such as the cæcum, the hepatic or splenic flexure, the sigmoid colon or the rectum. This cause is rare as compared with some others, yet the hope that it may be the one responsible prompts much of the therapy for acute obstruction, particularly the use of enemata, and there is no way to be sure it is not the cause until the attempt has been made to remove it and has failed.

### TREATMENT

The time comes when investigation must be followed by action, and this is the most difficult moment of all. It is easy to ask questions, to palpate, to percuss, to give an opinion, but it is hard to decide at last just what to do and not to do. In the maze of possible courses that the physician may pursue, it is essential that he have a few well-defined rules upon which he

can depend for guidance. Such are the following, whose value has been proved by experience.

1. Place the patient as soon as possible in a well-conducted hospital, where careful observations can be made of the temperature, pulse, blood, urine and other details that tell from hour to hour how the case is progressing, where the procedures indicated in treatment can be properly carried out, such as lavage, enemata, intravenous injections and abdominal applications, and where all is ready for laparotomy, if this eventually becomes advisable.

2. Give no food whatever after the diagnosis is made and as little water as possible. Meet the need for fluids by hypodermoclysis with normal salt solution or by intravenous injection of this or of 5 or 10 per cent. glucose solution.

3. Give no cathartic drugs. They only increase the patient's suffering and may even lead to a worse condition than that for which they are administered.

4. Give no opium in any form. It adds bowel paralysis to bowel obstruction, decreases elimination of poisons by skin and kidneys, and blunts the patient's sensibility to pain so that he is given a false sense of improvement.

5. Wash out the stomach with warm bicarbonate of soda solution, one teaspoonful to a pint of water, repeated as often as needed to check vomiting. This not only gives comfort but decreases toxæmia.

6. Use enemata at the outset and repeatedly, every few hours; of warm olive or cotton seed oil; of milk and molasses (one part of molasses, two parts milk, injected warm); of Epsom salts two ounces (60 grams), glycerine two ounces (60 c.c.) and water to make one pint (500 c.c.); of plain soap-suds; or of soap-suds with one teaspoonful (4 c.c.) of spirits of

turpentine to the pint. But if enemata have accomplished no results in twenty-four hours, they probably never will; if not after forty-eight hours, their use ought to be abandoned.

7. Apply hot compresses to the abdomen of flannel or wool soaked in hot water, or turpentine stupes, made by sprinkling turpentine on the hot compresses, or hot poultices of flaxseed meal, or hot antiphlogistine or some similar mixture of clay and glycerine such as the *cataplasma kaolini* of the pharmacopœia. All these give the patient comfort, and they may relax spasm of the bowel sufficiently to overcome obstruction.

8. Call a surgeon in consultation early, so that he may share in the responsibility for the decision as to when medical efforts shall cease and surgical shall be employed, and so that, if the ultimate outcome is unfavorable, the charge can not be made that the surgeon was called too late.

## CHAPTER XXVII

### CHRONIC INTESTINAL STASIS

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Constipation is one of the most common of all human ailments, and yet it is almost impossible to secure agreement among physicians as to just what is meant by this term, what causes the condition, what are the effects, or what to do about it. The truth seems to be that there is considerable individual variation as regards bowel habit, so that what is normal for one is not for another. The majority seem to find one free evacuation of the bowels each day essential to their well-being, but there are those who regularly have two and are not comfortable with less, while others have no complaint to make if defecation takes place only every other day or at even a longer interval. The only way to decide, therefore, what constitutes constipation, is not by the number of stools but by the effects upon each person of their own bowel elimination, and no rules can be made to apply to all. The decision must rest upon whether the individual maintains good health and efficiency, whatever the habit in this matter, and presents none of the complaints that can rightly be attributed to chronic intestinal stasis.

Properly classified, true constipation should always be regarded as a disturbance of function, not as the result of any recognizable organic disease. A number of such diseases of the gastrointestinal tract cause irregular and inadequate bowel movements as one symptom, but in such cases a diagnosis of chronic intestinal stasis is not justified. The functions of the intestine, disturbance of which is responsible for chronic constipation, are motility, by which the contents are kept moving along, and sensation, by which consciousness of the contents leads to desire for their expulsion. If motility is diminished and sensation is blunted, evacuations are bound to be irregular and inadequate. The factors that lead to decreased peristaltic power are first of all the prevalent habits of eating and drinking, too much food at meals, food with too little residue, food taken too frequently during the day and night at irregular hours, too little water. Second in importance come lack of exercise, sedentary occupation, over-use of the automobile in getting about, idleness and in general failure to keep the abdominal viscera agitated at least a part of the time. Third, exhaustion of nervous energy by mental overwork, or anxiety or worry; in general the condition known as neuræsthenia, however produced, is an important factor, because it causes faulty supply of power to the intestinal wall. Disturbance of sensation in the bowel concerns particularly the rectum and is the result of habitual lack of observance of inclination to empty it, because of fancied lack of time, or because of modesty or of the absence of facilities when the desire comes. Such repeated disregard of Nature's call leads gradually to diminution of the normal impulse and loss of the normal expelling power, when the time at last arrives that is convenient and opportunity is afforded that proves satisfactory.

## CLINICAL HISTORY

Disturbances of health of all sorts and kinds have been attributed to abnormal bowel elimination. Some have been charged to it without adequate proof, but there are others that seem to be associated with it so frequently as to justify the conclusion that they are related as cause and effect. *First* among these comes habitual interference with the stomach's functions, such as loss of appetite, sense of heaviness, fulness and discomfort after meals, early satiety and belching of gas. These symptoms all indicate loss of motility. Whether this disturbance is reflex, the result of a message sent back to the stomach to discourage further introduction of food into a bowel that already fails to care properly for what it has received, or whether it is the result of mechanical stasis, as the blocking of a sewer at any point results in stagnation far back in its course, or whether it is accounted for by chemical influences, toxins absorbed from the stagnant intestinal contents entering the blood in excess and then becoming excreted vicariously by the gastric mucous membrane as well as by other avenues of elimination, all these explanations have been offered, but for none of them is there scientific proof. The fact remains, however, that chronic intestinal stasis frequently is associated with the symptoms that indicate disturbance of gastric motility.

*Second*, the large bowel, like any other sewer, is an unclean place, containing food refuse that has undergone decomposition and putrefaction together with dead desquamated cells and myriads of bacteria. Poisons, that are continually manufactured here as a result of chemical changes, are routinely passed on to the liver by the portal vein to be destroyed by this organ as one of its special functions. This detoxicating power of the

liver is one of the chief safeguards to health in every individual. If this power becomes impaired by overwork, if too many toxins are constantly carried to the organ because they are not properly eliminated by defecation, then some of them are allowed all the time to pass on into the blood. Thus develops the symptom-complex known as "auto-intoxication" or "biliousness". The characteristic complaints assigned to this are a habitual sense of weariness, indisposition to exertion, sluggishness and inertia, dizziness and dull headache, depression of spirits, inability to concentrate the mental faculties. These develop along with the other symptoms already mentioned, indicating impairment of gastric motility, together with others next to be described, of a more disturbing character.

*Third*, pain in the head is a frequent consequence of chronic intestinal stasis. This may take the form of a hemicrania with nausea and prostration. The patient awakens in the early morning with such an attack, and it persists for a day or a day and a night, through one eye and one side of the head, usually violent and incapacitating. Patients learn for themselves that such a headache disappears more quickly if a brisk purge is taken that clears out the bowel. It also seems to be true that in some instances at least typical attacks of migraine, characterized by onset with flashes of light and blurring of vision followed by violent pain in one side of the head persisting for an hour or two, may be due to faulty bowel elimination and may be avoided, if the intestinal tract is regularly kept free from accumulation. Finally, there is a low, dull, nagging type of headache, present more or less every day, resembling that produced by eye-strain, when there is an uncorrected error of refraction, quite characteristic of faulty bowel elimination and made to disappear by daily evacuations.

*Fourth*, there is a dirty, earthy, sallow pigmentation of the skin in chronic intestinal stasis that is always suggestive, even if not diagnostic. Mixed with this at times are brown patches or spots over the face, neck, hands and arms. Acne also is a frequent complication. Bad-smelling perspiration, particularly from the axillæ, is another common associate of faulty bowel elimination.

*Finally*, these patients have much to tell about the amount and character of the bowel discharges, which they constantly make it their duty to observe. They complain that there is no daily evacuation, or if there is, the stool is small, scrappy and inadequate. Constant investigation of what is passed, as regards the consistency, color, shape and even the odor of the excrement becomes a habit with these patients. This occupation gradually becomes an obsession with them until all their daily activities hinge upon the behavior of their bowels.

### PHYSICAL EXAMINATION

This confirms the patient's own observation as regards poor complexion, offensive breath, disagreeable odor of the perspiration and coated tongue. The blood pressure usually is low, the systolic averaging 110 or less. But no abnormalities are found over the abdomen to indicate the presence there of any serious organic disease. There may be a succussion splash over the stomach, palpable doughy masses in the course of the ascending colon and a packed rectum due to retained fæces. But there is no definite tenderness found anywhere, no tumor mass, no sign of any cause for the slow and inefficient peristalsis. This absence of physical signs, excluding organic disease and indicating that functional disorder is responsible for the symptoms, is highly important in diagnosis. But in every physical exam-

ination routine digital exploration of the rectum should never be forgotten.

### LABORATORY EXAMINATIONS

Complaints about the stomach form so large a part of the story told by patients with chronic intestinal stasis that only by *gastric analysis* can the truth be discovered as to what these symptoms really mean. There is no characteristic analysis to be expected in these cases. Secretion may be within normal limits, increased or decreased. Even when free HCl is much below normal average, there is not found the excess of mucus that indicates chronic gastritis. The significant discoveries made by gastric analysis are those that indicate disturbance of motility rather than of secretion. The existence of atony is shown by finding in the fasting contents fragments of food retained from the previous meal and after a test meal a larger amount of contents than usual when extractions are made with poor trituration of such a meal as the Ewald. Such evidences of faulty motility in the stomach are not always found with chronic intestinal stasis and may be found in a number of other conditions besides, but when they are discovered, they afford an adequate explanation for the symptoms referred to the stomach, when chronic constipation furnishes the main part of the story.

The *stools* frequently are scanty, hard and dry, in cylindrical form or else in small, round lumps like the excrement of sheep. But they show no mucus of consequence, unless chronic colitis is associated, no blood or pus suggestive of neoplasm, no ova or parasites, to explain the disturbance of intestinal function. The *urine* is normal except that it frequently contains indican. The value of this as a sign of chronic retention of fæces in the bowel is not as much emphasized now as it used to be some

years ago, but it still is of significance in connection with other evidence and should not be discarded. The *blood count* may show a secondary anæmia but never to any degree from chronic constipation only, and the icterus index is not above normal, in spite of the patient's suspicious color, unless some complication exists.

### X-RAY EXAMINATION

The chief value of this is to eliminate organic disease of the gastrointestinal tract that might be responsible for inadequate bowel elimination. After a barium meal the stomach shows no defect or irregularity of contour nor any sign of pyloric obstruction in spite of its disturbed functions, and though it may empty slowly, it does not retain food. After a barium enema the large bowel likewise shows no signs of organic disease, such as chronic appendicitis, or diverticulitis, or neoplasm that might account for the obstruction. Ptosis of the transverse colon or redundancy of the large bowel, occasionally demonstrated by x-ray films, may or may not play a part in causing delay in the passage of contents.

### DIAGNOSIS

It is not difficult to conclude that a patient suffers from chronic intestinal stasis, because the patient's history and a few days of observation will prove beyond question whether bowel movements are adequate, no matter what the other symptoms may be. The attempt to distinguish various forms of chronic constipation, such as atonic and spastic, seems to be more academic than practical and to accomplish no good purpose. The most important question in every case, however, is whether faulty elimination is only a symptom of some underlying or-

ganic disease or is purely a functional disturbance. To answer this it is necessary to review the history, physical examination, laboratory findings and x-ray films for the evidence they may afford and all forms of organic change must be excluded before the responsibility for delay can be rightly charged to atony of the bowel or to blunting of sensation in the rectum.

As regards symptoms attributed to the stomach there is no characteristic history to prove that its chronic disturbances of function are due to intestinal stasis. Even for disagreeable odor of the breath or for bad taste in the mouth, an explanation should be sought in pyorrhœa or decayed teeth or plugs of decomposed detritus in tonsillar crypts or purulent accumulation in a sinus, before they are assumed to originate from the stomach as a result of chronic constipation. All other details commonly related by these patients are best explained by atony, which may be a part of a general slowness of peristalsis throughout the entire gastrointestinal tract.

Headaches are not always due to retention of fæces in the colon. But the periodic attacks that come on overnight, with which the patient wakes in the morning, commonly involving only one-half of the head, especially intense in one eye or one temple, lasting for a day with nausea and a sense of prostration and incapacity for duty, and disappearing after the bowels are freely moved, should always suggest an intestinal origin. When headaches of this character recur repeatedly in individuals, who not only have constant difficulty in getting their bowels to move but also show such peculiarities as poor nutrition, pigmentation of the skin and low blood pressure; they suggest chronic poisoning.

It can not be denied that the tendency is to place too many cases of constipation in the functional group without proper or

thorough search for some organic disease that causes it. But neither can it be denied that many patients are seen in whom no such cause can be found, no matter how much time and care are given to their investigation. These are the patients to be properly placed in the group called chronic intestinal stasis and to be treated accordingly.

### TREATMENT

Three of the minor ailments of humanity, about which the advertising pages in the popular magazines are much concerned, are bad breath, odorous axillæ and poor complexion. All of these are better controlled by keeping the bowel clear than by any of the local applications so widely heralded. Long continued disturbances of gastric function, persistent in spite of all sorts of treatment directed to the stomach, now and then disappear as soon as the colon is persuaded to perform regularly and completely its function of eliminating waste. Headaches that recur at inopportune moments and upset the plans made for the day not infrequently cease to annoy as soon as proper attention is paid to preventing stagnation of intestinal contents. Finally, not only may mental capacity be decreased but even moral sense may be impaired by the constant retention of impurities in the bowel and their absorption into the blood. Thus the treatment of chronic intestinal stasis becomes a problem of interest not only to the individual afflicted but to the community in which he lives, and thus to the happiness and usefulness of numerous other people.

The purpose of treatment is to increase the muscular power of the colon wall by every possible means. Stimulation to overcome its sluggish action is obtained by diet, by exercise, by massage and other physical aids, and by medicinal remedies.

(1) *Diet.* — The diet most often useful in chronic intestinal stasis is one containing sufficient residue, after digestion and absorption have occurred, to induce peristalsis by its bulk. Many of these patients are afraid to eat, because their stomachs annoy them and their bowels do not move satisfactorily, but by failing to take food of the right kind and in sufficient quantity they simply perpetuate their trouble. The following diet list has long been advised for this group of people, with satisfactory results:

*I. Foods that should be eaten freely and constitute a large part of the diet*

1. *Fresh vegetables:* (a) raw, as lettuce, tomatoes, cucumbers, celery, radishes; (b) cooked, as spinach, cauliflower, cabbage, sprouts, green peas, string beans, carrots, parsnips, turnips, egg-plant, asparagus, artichoke, onions. 2. *Fresh fruits:* raw, or cooked without sugar; as peaches, apricots, berries of any kind, apples, grapes, figs, plums, nectarines, pears, melons, cherries; when the fresh fruits can not be obtained, citrus fruits, as oranges or grape fruit; or stewed dried fruits, as peaches, apricots or prunes, cooked without much sugar; orange marmalade. 3. *Coarse cereals:* as bran, whole wheat, graham flour, corn meal, oatmeal, Roman meal; cooked as a mush or made into bread or muffins; eaten with cream or plenty of butter.

*II. Foods that should be eaten sparingly, always in moderate amount*

1. *Meats:* never to be taken more than once a day and then only in small quantity; mutton or lamb, in roast or chops; beef, in roast or steak; chicken; turkey; freshly cooked tongue; liver; brains or sweetbreads. 2. *Fish:* any kind, if taken in place of meat and not as an extra course at the same meal; but only fresh fish, not salted or smoked. 3. *Shell-fish:* oysters may be taken served raw or cooked as desired; but not crab, lobsters or shrimps. 4. *Eggs:* not to be taken every day and only as a meat substitute when taken at all; cooked in any way desired. 5. *Desserts:* Baked or stewed fruits; puddings made of corn-meal, sago or tapioca with figs, prunes or apples.

*III. Foods that should be avoided*

1. Among *meats*, all forms of pork, either fresh, salted or smoked (including ham or bacon); veal; all potted and preserved or dried meats; wild duck. 2. Among *fish*, all salted, smoked or canned varieties (such as sardines, mackerel, herrings, codfish, canned salmon etc.), crab, lobster or shrimps, whether canned or fresh. 3. Among *cereals*, all new bread, hot cakes, waffles, cakes and pastries, rice, Italian paste. 4. Among *beverages*; milk (though cream and butter may be taken freely, with cereals or vegetables); tea; wines; ice-cream sodas.

Sometimes simple additions to the diet accomplish wonders. The morning cup of coffee with or without cream acts for many as a sufficient stimulus to peristalsis to produce the desired after breakfast bowel evacuation. For others the proverbial "apple a day" works better as a laxative than does any drug. For still others it is an orange or a pear, or prunes or figs, taken regularly at the morning meal, that best accomplishes the purpose. Some find in Roman meal the remedy they require, some in bran, some in Scotch oatmeal. For nearly every one a valuable aid to peristalsis is a glass of water, hot or cold, taken on rising each morning, at least a half hour before breakfast. There is no one food or group of foods that avails for all. But even so, the list recommended furnishes a proper basis and a suitable guide.

(2) *Exercise* is almost indispensable to health. All parts of the body suffer from a sedentary life that never brings the muscles into active and general use. But certain forms of exercise are particularly valuable to assist the digestive organs and especially the intestines in the performance of their functions. These exercises include outdoor sports such as walking, tennis, golf, swimming, horseback riding and rowing. If these methods are prevented by inclement weather, or if the patient can not be

persuaded to make use of them regularly and systematically, then indoor exercises can be substituted; but they too are not of much avail unless carried out persistently. Simple devices, especially suited for the patient with chronic intestinal stasis, because they strengthen the abdominal muscles, are the following: (*a*) lying on the back, raise and lower the right leg five times, keeping the knee unbent, (*b*) then the left leg in the same way five times, (*c*) then both legs together five times, (*d*) then standing erect, with hands raised above the head, bend the body forward at the hips until the fingers touch the floor, then resume the erect posture, repeating these movements five times. These exercises need not take more than ten minutes each morning, before rising and immediately after, and if they are kept up faithfully, they can be depended upon to give added power to weak abdominal muscles. It is to be remembered in advising exercise of any sort that for older and debilitated individuals the fatigue incident to it may counteract the effects sought from it; for such patients active exercise should not be advised.

(3) *Massage* of the abdomen each day by a professional masseur undoubtedly is of benefit. But patients who can not afford the time or the money that this kind of treatment costs, can do the same thing for themselves in a crude but fairly satisfactory way without much loss of time. They are instructed to make pressure along the course of the colon, from the lower right quadrant upwards, then across about the level of the navel, then down again on the left side, repeating this procedure a dozen times; or to make more vigorous kneading movements over this same course; or to employ a small chamois-covered iron cannon ball and roll it along over the same route for ten or twelve rounds. Other physical aids of proved value in restoring tone to

enfeebled abdominal and intestinal walls are the morning cold bath, by shower or plunge, followed by vigorous friction of the body with a Turkish towel; or hydrotherapy systematically administered by douche or spray to the abdomen itself and up and down the spine.

(4) *Drugs* should be the last resort but in many cases their assistance is required. If chosen wisely, the harm they do is not to be compared with that which comes from chronic intestinal stasis, and though they are rightly looked upon as an evil, they are the lesser of two evils. Those, who resent taking laxatives because they object to establishing a habit, should be reminded that they already have a worse habit, of letting their bowels go without proper and regular evacuation, and those who argue that medicinal aid is unnatural must remember that their loss of intestinal function is likewise unnatural, and that defects require what normal bodies do not. Of all the long list of laxatives available, the most beneficial and least harmful for repeated use is some form of cascara sagrada. This can be best prescribed as the fluid extract in dose of fifteen to thirty drops, or as the dry extract in a tablet of three or five grains (0.2 to 0.3 gm.) at bedtime. A useful formula prescribed for years past with satisfactory results is composed of fluid extract of cascara and syrup of tolu of each two ounces (60 c.c.) with oil of anise ten drops; of which the average dose is one teaspoonful (4 c.c.) at bedtime, increased or decreased as required. After all, the patient who uses cascara as a laxative is taking only the juice extracted from the bark of a tree, and if he could get the same result from the daily swallowing of a juice from some fruit, there would be no feeling of drug-taking. Yet the one kind of vegetable extract is as harmless as the other, only more stimulating to peristalsis.

As regards other popular remedies for constipation, the great number of laxative pills of various formulæ offered to the profession by the drug manufacturers testify to the frequency with which such assistance is sought. They all have their virtues and most of them are mild enough in their action so that their persistent use does no harm. But it is better if possible to obtain the desired result by simpler means. It is certainly unwise for the patient to select for frequent use such a drastic agent as the compound cathartic pill or others resembling it. Many different preparations of mineral oil (liquid petrolatum), either alone or combined with agar, at present are advertised extensively. Their main recommendation is that they are harmless. For some patients they act well, but for others they act not at all. Their range of efficacy is never as wide as that of the extract of cascara, fluid or dry. The salines are not advisable for regular use, valuable as they are in an emergency, for they produce their effect by causing accumulation of fluid in the intestinal lumen, mechanically distending the bowel and so inducing the desire for their expulsion. But frequent repetition of this process weakens the bowel wall.

(5) *Enemata*. — For those who hesitate about taking any laxative by mouth, there remains the alternative of stimulating daily evacuations by injections into the rectum, not for absorption but for mechanical effects. By this route the most useful remedy for regular employment is oil in some form, preferably olive or cottonseed. The following written instructions are given to patients as to the proper method of managing these injections, and have been found satisfactory after a number of years of trial. But they are much more troublesome to follow and less apt to be carried out faithfully than the advice to take each night a laxative tablet or mixture.

“A small glass funnel and a large size soft rubber catheter are to be obtained from the drug store and the catheter attached to the funnel. The patient is to lie on the back or the left side. The tube is to be lubricated with vaseline and passed into the rectum about eight inches. Then six ounces of warm olive or cottonseed oil are to be poured in slowly and the tube withdrawn. This should be done after the patient is ready for bed, and the object is to have the oil retained all night. If the oil is rejected at once or leaks out during the night, a smaller quantity should be used next time. It is best to have a napkin worn to avoid soiling the bed linen. The following morning, after breakfast, the patient is to go to the toilet and remain there until a bowel movement results or until fifteen minutes at least have elapsed. If no result has been obtained at the end of fifteen minutes, a glycerine suppository should be inserted as an additional stimulant. This procedure should be carried out regularly each night and morning for at least two weeks, then every other night, then only twice a week until a regular habit is established.”

The morning enema of soapsuds or of plain water is not advisable for regular use, because it acts by stretching the rectum which thus ultimately becomes weakened and incapable of exercising its normal function. But for emergency use no remedy is more reliable, and no harmful result need be feared unless it is continued day after day for months.

(6) *Psychotherapy*. — Finally, patients with chronic intestinal stasis always need encouragement. They are afraid to abandon strong cathartics they have adopted. They hesitate to try new methods. They have lost confidence in the working of their own bodies, and their mental and moral stamina has degenerated with their physical. The fear that the bowels will

not move without artificial aid itself restrains their normal action, and systems of psychotherapy that remove this fear, no matter under what name they work, frequently perform miracles for these individuals. With proper diet, healthful exercise and mode of living, and with a mind freed from fear and filled instead with confidence, the use of laxative drugs undoubtedly can be reduced to a minimum and in some cases dispensed with altogether.

CHAPTER XXVIII

INTESTINAL PARASITES

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What are commonly known as “worms” are not commonly observed in patients on the Pacific Coast and less frequently now than twenty or more years ago, because of better inspection and control of food and drink by the Public Health Service. There are only a few varieties seen in ordinary practice. There are microscopical parasites such as the *Entamæba histolytica*, already considered in a special chapter devoted to the effects it produces, and the flagellates such as giardia and chilomastix, which have never been generally accepted as pathogenic agents. But there remain certain parasites large enough to be seen by the naked eye and of a form to justify their inclusion under the general designation “worms”. These are pin worms, round worms and tapeworms.

PIN WORMS

Pin worms are known also as thread worms and seat worms as well as by their scientific title *Oxyuris vermicularis*. These

are observed more often in children than in adults but may be found at any age. Their eggs enter the intestinal tract in food or water or from the hands of infected persons. They develop and multiply in the large bowel and as far up in some instances as the cæcum and appendix but most often in the rectum. From this site they crawl out the anus and cause itching there, or in the female may cross the perineum and invade the vulva and vagina. They are particularly active at night when the patient is warm in bed and may then cause itching intense enough to disturb sleep. The only proof of their presence is their discovery either in the stools or in the folds of the mucous membrane about the anus or vulva. They are small and white, about a quarter to a half inch long and resemble bits of white sewing thread, from which peculiarity they derive one of their names.

These little parasites usually are destroyed most satisfactorily by an infusion of quassia chips, made by steeping for a half hour one ounce of the chips in a pint of boiling water. After the rectum has been thoroughly washed out with warm borax solution, one tablespoonful to a pint, about six or eight ounces of the warm quassia infusion should be injected into the rectum at bedtime to be retained overnight. This must be repeated once each twenty-four hours for at least a week, and then every other night or twice a week for at least a month. Itching about the anus leads to scratching, eggs of the parasite may thus be introduced under the finger nails and then when food is taken again it may be contaminated by handling and thus re-infection may occur. To prevent this pajamas or tight drawers should be worn at night to prevent direct contact of the fingers with the infected area, and finger nails should be cut short. Persistent care about details will always succeed

ultimately, but the parasites are not easily made to disappear. In the writer's experience no drug given by mouth has ever succeeded so well as the quassia infusion by rectum.

### ROUND WORMS

Round worms, scientifically designated *Ascaris lumbricoides*, are likewise more often seen in children than in adults but may be observed at any age. On the Pacific Coast more cases apparently are seen in Orientals, such as Japanese, Chinese and Filipinos, than in white children. The eggs of this parasite are taken into the body with contaminated water or with uncooked vegetables. They develop into a worm five to ten inches in length, yellowish or white, resembling an angleworm. They live in the small intestine and usually are few in number. So long as they remain at the site of their original development, they cause few or no symptoms, and their presence in the bowel is rarely suspected until they are discovered in the stools. But they have a tendency to travel and so may make their way into localities where they cause more trouble than in the bowel itself. A round worm has been known to make its way into the stomach, then up the œsophagus and out the mouth or into a bile duct, where it caused obstruction, or into some other unusual situation. Exceptionally, when numerous, they may tangle themselves into a ball large enough to obstruct the bowel. But usually they cause so little disturbance that their ultimate passage in the stool comes as a distinct surprise.

To effect the removal of this parasite, in case its presence is suspected or in case one worm has been passed already and it is considered probable that others remain behind, the specific remedy employed for years past has been santonin. This should be combined with calomel, about one to three grains of each,

the amount depending upon the age of the patient, given in one single dose, at bedtime or in several small doses at short intervals. This should be followed the next morning or one hour after the last powder or capsule by citrate of magnesia or Epsom salts. Success almost invariably follows this plan. If worms are passed after this treatment but doubt remains whether all have been removed, a second course may be given after a few days or a week. If no worms are found after this second course, no further treatment is indicated, but if more do appear, a third course should be given after a week has elapsed. But santonin in too large or too frequent doses may produce disagreeable and at times serious effects and should not be administered without caution.

#### TAPEWORMS

Tapeworms are parasites of considerably more importance. The scientific name for them is *Tænia*, and while there are a number of different varieties recognized and classified, there are only three that are encountered with any frequency in clinical medicine. These are (1) *Tænia saginata*, or beef tapeworm, (2) *Tænia solium*, or pork tapeworm, and (3) *Diphyllobothrium latum*, or fish tapeworm. None of these parasites are common where health authorities carefully inspect food supplies, as in most parts of the United States, or where meat and fish are well cooked before being eaten. What enters the patient's bowel in this instance to start the parasite's development is not the egg but the tiny cyst-like larval form in the muscle of beef, pork or fish taken as food. One of these fastens itself to the wall of the small intestine and grows from this point pushing out segment after segment farther and farther away from the head or site of beginning until ultimately the parasite

may reach a length of 18, 20 or even 30 feet before its presence is discovered, and its removal is effected. The beef worm is the one that has been observed most frequently in this country, the pork worm second and the fish worm, though common in Europe, is rarely or not at all observed in the United States, except in patients who have brought the parasite with them from Finland or some other country around the Baltic where the fish used for food are commonly infested or in certain parts of the country, as in Michigan, where native fish are showing considerable infestation.

### *Clinical History*

Tapeworm in the bowel may cause no symptoms at all, and its presence may not even be suspected until segments are passed in the stools. The classical symptom, long considered diagnostic, is a large appetite, but this does not mean much by itself. It may never form part of the patient's story even when tapeworm is present, and it may accompany a number of other ailments without intestinal parasite to explain it. A little more significant is complaint of gnawing, nagging pain in the upper abdomen with a sensation of faintness, nausea and sinking coming on in the early morning on waking or during the day several hours after a meal. Some patients describe also a sense of commotion in the intestines, an uncomfortable squirming and twisting of the bowels, not painful but most disagreeable. This peristaltic unrest also is most likely to occur sometime after food is taken or in the early morning before breakfast, but usually it goes away entirely after a full meal. The bowel function as a rule is abnormal, but the stools may be either too few or too many. Reflex disturbances of the nervous system result in some patients, such as rest-

lessness, nervousness, irritability, poor sleep, dizziness, headache and rarely attacks of petit mal. But none of these symptoms are seen so regularly that they become diagnostic, and there may be none at all.

Sooner or later, whether symptoms have preceded or not, segments are passed in the stool and attract the patient's attention; sometimes an even more surprising proof of the presence in the bowel of a tapeworm is afforded by segments that wander out through the anus, during the day or night and are found in the patient's clothing or in the bed. The segments passed in the stool may be single, or there may be several in a chain or a portion of the worm a foot or more in length may escape with a bowel movement. Freshly discharged segments, while still warm, may show visible movement.

It is by seeing and identifying segments and by this alone that the diagnosis of tapeworm is reliably made. Stool examination is, therefore, essential to recognition of the presence of this parasite. If patients find objects in the feces that seem to them to mean tapeworm, treatment is never justified until the worm has been identified by the physician in the stool passed, or at least that portion of it containing objects supposed to be segments. Frequently such objects are simply bits of undigested food, or mucus casts or shreds, or such unusual articles as melon seeds. The true segment, flattened between two glass slides and held to the light, shows distinctly a structure that is not only characteristic of tapeworm but serves to distinguish one variety from the other. The portions of the worm thrown off with the stool are the oldest because farthest from the head and differ materially from the more delicate pieces nearer the point of origin. The mature segments of *Tænia saginata* are longer than those of other varieties and in their internal structure

show as many branches on each side as twenty to thirty, coming off from a median canal. The segments of *Tænia solium* as passed in the stools are shorter and relatively broader, with only seven to ten lateral branches on each side. The segments of *Diphyllobothrium latum* usually are square or broader than they are long. Thus it is possible not only to identify with certainty the character of the object discharged from the bowel but also to determine the kind of parasite that produced it.

The eggs produced by these parasites are found in the stools and are supposed to aid in diagnosis, even without the discovery of segments. Each variety has its own peculiar ovum, but it requires an expert laboratory technician to recognize them and to distinguish one form from the other. It is certainly not justifiable to act hastily upon the information furnished by eggs alone. If they really mean tapeworm, segments will sooner or later be passed, and until they are, it is wiser to postpone drastic treatment required for expulsion of the entire parasite.

### *Examinations*

Other methods of investigation are of little value compared with inspection of the stool. *Physical examination* gives no direct evidence that helps to make the diagnosis. *Gastric analysis* frequently shows hypersecretion produced reflexly, but does not tell where the pathological disturbance lies that caused it. Taken with the history, sometimes elicited, of distress in the upper abdomen coming late after meals, hypersecretion is more likely to be interpreted as meaning gastric or duodenal ulcer than tapeworm. But normal secretion or even hyposecretion may be found, even when tapeworm is present. The

patient's blood count may be reduced by the presence of the fish tapeworm in the bowel to a degree of anæmia resembling that of the primary form. This point is of importance when an obscure anæmia is encountered for which no cause at first is apparent. *X-ray examination* aids in eliminating other causes of digestive disturbance, such as gastric or duodenal ulcer, gall-bladder disease and chronic appendicitis, but gives no direct sign that proves tapeworm is present in the bowel.

### *Diagnosis*

Thus it becomes evident that neither history nor physical examination nor stomach analysis nor x-ray films, either alone or in combination, are sufficient to justify the diagnosis of tapeworm. The proof of its presence in the bowel depends upon stool examination and upon that alone. But this examination must be made by the physician himself or by some laboratory expert to whom the specimen is submitted. The patient's report about it must never be accepted as a basis for action.

With a history of vigorous appetite, with pain and nausea appearing several hours after meals or in the night time and relieved by taking food, with negative physical examination except possibly for epigastric tenderness and with gastric hypersecretion, the temptation is sometimes strong to diagnose *ulcer*, even though the x-ray report shows no defect in stomach or duodenum. This error is made occasionally, but it can always be avoided, if stool examination is thoroughly carried out. It is humiliating, after diagnosing and treating ulcer, to have the patient return one day with a stool showing tapeworm segments, which prove the original opinion entirely incorrect. No clinician cares to have this experience more than once.

If there is constant disturbance of intestinal functions, such as irregular bowel movements, constipation alternating with diarrhoea, cramps or at least frequent discomfort from peristaltic unrest, and if with this history tenderness is found over the cæcum, it is natural to suspect the *appendix* as the focus from which these various symptoms arise, even though no very decided evidence of pathological disturbance there is given by x-ray films. The stools again afford the clue to the correct diagnosis. Even if they are negative on repeated search on different days after cathartic medication, it is nevertheless wiser to delay operation for chronic appendicitis until further time has elapsed and further opportunity for stool observation has been given.

The highly nervous, excitable and introspective patients, set down as neuræsthenic or psychæsthenic, those with recurring headaches and bilious attacks, those with epileptiform paroxysms or chorea are all entitled to careful stool examination for evidences of tapeworm before they are relegated to the group of maladies for which no explanation can be found. Likewise patients with severe anæmia may be the victims of toxæmia from tapeworm and may rapidly regain normal hæmoglobin and red cells after their parasite has been expelled.

A man, aged 35, complained of what he called "nervous indigestion", which he attributed to anxiety about the lengthy illness of his wife. His disturbance of health was characterized by a variable appetite, nausea after taking food, an acute pain in the upper abdomen at times, described as "a deep-seated ache" and soreness to touch over the region of pain. Often at night he had a vague ache in his abdomen, and he arose each morning with a gnawing sensation in his stomach persisting until he ate breakfast. The bowels were constipated, and the anus itched. The patient was a large, well-nourished man apparently in good health and showing no signs of

organic disease in abdomen or elsewhere. After an Ewald test meal the total acidity was 60 and the free HCl 36. The urine was normal. At this stage in the investigation the patient went away on a hunting trip and noticed his feces for the first time because they were deposited on the ground. To his horror he observed in the fresh bowel discharge living and moving objects. Some of these he brought soon afterwards for examination, and they proved to be segments of tapeworm. On routine treatment to be described he passed the entire worm and the head. Careful measurement showed the parasite was over 30 feet in length. After this all previous symptoms disappeared.

### TREATMENT

As soon as possible after the presence of a tapeworm has been recognized, an attempt should be made to evict the unprofitable tenant. But this process is not so easy, and the history the patient gives frequently includes the account of several unsuccessful attempts to dislodge the worm. Sometimes these frustrated efforts have been made by the patient without competent advice, but sometimes by the physician, who has employed measures recommended as competent and generally approved by medical authorities. Even so the worm has remained to scorn the assaults made upon it in its home. By such histories as well as by personal experience the lesson is taught that the expulsion of a tapeworm from the intestine is neither a simple nor an easy task.

To succeed in treatment, a definite plan must be followed, and this should be carried out in a hospital, if possible, where all details can be properly supervised. But whether there or at home, the first step is to prepare the patient; the second is to give the remedy that will kill the worm; the third is to effect the expulsion of the worm from the body; and the fourth and

last is to overcome the irritation of the bowel caused by the drastic measures previously required. (1) On the first day the patient should be made ready by clearing out the bowel, by one ounce (30 c.c.) of castor oil, followed by a soapsuds enema; and by limiting the diet to liquids such as weak beef, mutton or chicken broth, plain tea or coffee, in amounts not to exceed six ounces (180 c.c.) every three or four hours. (2) On the morning of the second day, while still fasting overnight, the patient is given the drug selected to destroy the worm. Almost invariably a successful result follows the use of male fern, prescribed as the oleoresin of aspidium. The average dose of this is two drachms (8 gm.). It is best administered in capsules, each containing fifteen grains (1 gm.). One of these is given every ten minutes until all are taken. (3) Then follows the administration of some remedy to expel the worm, killed by the aspidium. For this purpose one ounce (30 gm.) of Epsom salts usually suffices. It should be given one hour after the last capsule. If no bowel movement has resulted in another hour, a cupful of hot broth or hot black coffee will assist by stimulating peristalsis. When the bowels move, the discharges should be received in a vessel containing a little warm water, and all that passes the anus at this time and subsequently for twenty-four hours should be saved carefully for inspection and examination, and careful search must be made for the head. (4) On the third day, following the vigorous medication described, the patient should remain in bed, take liquid diet only and receive twenty grains (1.3 gm.) of bismuth subcarbonate every two hours, until diarrhoea has ceased and abdominal tenderness has disappeared.

If the head is not found, even though a large amount of worm has been passed and fine segments have been seen that must have been near the head, a repetition of the treatment

should be delayed for at least three months or until segments are seen again in the stools. For the head may have been forced out but overlooked, and the only positive proof that it remained behind is furnished by the reappearance of segments after the worm has grown again sufficiently long to give them off. Before that time no specific treatment is justifiable. If it is decided ultimately to repeat the plan, because segments are once more being discharged, it is usually wiser to try for that particular parasite some other anthelmintic drug. The second choice should be Tanret's pelletierine, a preparation made in France from the bark of the pomegranate root. The contents of the little vial, which represent the dose required, are mixed with a small amount of sweetened water and taken in place of the capsules of aspidium while the patient is fasting. All other directions, before and after the pelletierine is swallowed, are to be the same as previously described.

If as a result of treatment only a few feet of coarse segments are obtained, the entire plan may be repeated in a few days after the first trial, using again the aspidium or the pelletierine or some other anthelmintic drug or combination of drugs. No one preparation succeeds in every case.



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